

**FINAL**

**Technical Report**

Development of a Relative Source Contribution Factor for  
Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX)

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## Executive Summary

This report presents the development of a relative source contribution (RSC) factor for hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX, or Royal Demolition eXplosive), a military munitions explosive. An RSC accounts for all sources and non-occupational exposures from RDX and apportions these amounts to each source so that an individual's total dose does not exceed the reference dose (RfD). The application of the Exposure Decision Tree approach (subtraction method) recommended by the United States Environmental Protection Agency (USEPA) enabled the selection of an RSC of 50% rather than the default RSC of 20%.

RDX is a synthetic chemical unknown to occur naturally, which limits its environmental occurrence. There are few environmental regulations, standards, or guidance values for RDX, but an increasing number of states are developing standards and guidelines for RDX. Data from reputable databases such as MEDLINE (PubMed), Defense Technical Information Center (DTIC<sup>®</sup>), and SciFinder were utilized to collect data required for the USEPA's Exposure Decision Tree approach. The data suggest that exposure to RDX is not anticipated to be a national exposure concern given that production and use of RDX is limited to restricted areas, such as artillery ranges and Army munitions plants; RDX has limited civilian use.

A conceptual exposure model was utilized that identified the relevant potential sources for a highly exposed receptor proximate to an area where RDX was released. Potentially contaminated media include soil, groundwater, and surface water. Potential exposure pathways include ingestion of soil, water, and contaminated local crops and fish, and dermal contact with soil and from water used in bathing. These pathways are limited to areas that are in close proximity to current or former military bases where RDX may have been released into the environment and are not applicable nationally.

The chemical and physical properties of RDX do not suggest widespread dispersion in the environment; RDX has low water solubility, slow dissolution in aqueous solution, low vapor pressure, and a low affinity for hydrophobic substances, which suggests that RDX would have limited retention in soil. Nonetheless, laboratory and field studies have demonstrated RDX's potential to leach from soil— it has a medium-to-high mobility in soil resulting in its potential to leach to groundwater. RDX is not very lipid soluble and likewise has a low potential to bioaccumulate in aquatic organisms, but plants have been shown to bioaccumulate RDX from contaminated soil and irrigation water.

Given the physical/chemical properties and the available environmental occurrence data on RDX only at current or former military sites, there is adequate reason to support a chemical-specific RSC term for RDX of at least 50% utilizing the USEPA process.

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## List of Acronyms

AAP	Army Ammunition Plant
ATSDR	Agency for Toxic Substances and Disease Registry
BAF	bioaccumulation factor
CAS	Chemical Abstract Service
CDI	chronic daily intake
DoD	US Department of Defense
FUDS	Formerly Used Defense Site
GI	gastrointestinal
HHS	US Department of Health and Human Services
HSDB	Hazardous Substance Data Bank
IRIS	Integrated Risk Information System
K <sub>ow</sub>	octanol/water partition coefficient
kg	kilogram(s)
m	meter(s)
m <sup>3</sup>	cubic meter(s)
mg/L	milligrams per liter
NFA	National Fireworks Association
ng	nanograms(s)
NIH	National Institute of Health
NIOSH	National Institute of Occupational Safety and Health
NLM	National Library of Medicine
NOES	National Occupational Exposure Survey
NPL	National Priorities List
PPIS	Pesticide Product Information System
ppm	part(s) per million
PUF	plant uptake factor
PWG	Pathology Working Group
RAIS	Risk Assessment Information System
RDX	hexahydro-1,3,5-trinitro-1,3,5-triazine (or Royal Demolition eXplosive)
RfD	reference dose
RSC	relative source contribution
SDWA	Safe Drinking Water Act
SSL	soil screening levels
UCMR	Unregulated Contaminant Monitoring Regulation
UK	United Kingdom
US	United States
USACE	US Army Corps of Engineers
USACHPPM	US Army Center for Health Promotion and Preventive Medicine
USAMRDC	US Army Medical Research and Development Command
US FDA	US Food and Drug Administration
USEPA	US Environmental Protection Agency

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# 1. Introduction

Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX, or Royal Demolition eXplosive) (Chemical Abstracts Service [CAS] Number: 121-82-4), also known as cyclonite or hexogen, is a synthetic chemical having both military and civilian applications as an explosive. It can be released into the environment by way of waste streams generated during the manufacture, use, and disposal of the pure product or RDX-containing munitions. Human exposure may occur during the manufacture of RDX or RDX-containing explosives or through contact with contaminated environmental media. RDX is of specific interest to the United States (US) Department of Defense (DoD) because of its extensive use in military munitions. There are potential impacts to the DoD mission resulting from the US Environmental Protection Agency's (USEPA's) ongoing health reassessment. In addition, the USEPA is also in the preliminary stages of developing a drinking water standard for RDX under the Safe Drinking Water Act (SDWA).

The purpose of this report is to develop a relative source contribution (RSC) factor for RDX. The USEPA calculates an RSC as part of deriving drinking water health advisories and drinking water standards. The RSC is meant to ensure that when populations are exposed to a chemical from multiple sources, the total exposure will not exceed the reference dose (RfD) (USEPA, 2000). RSCs are an attempt to account for all sources and routes of non-occupational exposures. They are calculated for chemicals that are non-carcinogens or threshold carcinogens.

As demonstrated in this report, exposure to RDX is not widespread and there are adequate data available to move from the default RSC of 20% recommended by the USEPA (USEPA, 1989). There may be site-specific situations where populations are living near current or former military installations that produce or use RDX-containing materials and may be exposed to some level of RDX. The USEPA encourages states and authorized tribes to make alternative exposure and RSC estimates based on local data. These local situations would be evaluated on a site or local basis. This report uses the USEPA approach, which may be useful for calculating an RSC for a local situation.

## 2. Background

In 2000, the USEPA developed RSC guidance for assessing total human exposure to a contaminant and apportioning the RfD among the medium of concern (USEPA, 2000). The RSC is a factor used for risk assessment of chemicals in drinking water to allocate only a portion of an individual's total intake of a non-carcinogen to drinking water. This factor is applied to indicate that a chemical present in drinking water may also be present in other media to which people are exposed, such as air, food, and soil. The RSC ensures that the level of a chemical allowed by a criterion, when combined with other identified sources of exposure common to the population of concern, will not result in total exposures that exceed the RfD. The RSC for a chemical is derived by application of the Exposure Decision Tree approach (Figure 1) published in the USEPA's *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health* (USEPA, 2000). The approach considers the adequacy of available exposure data, including relevant sources and media of exposure, to support the determination of an appropriate

RSC value. This approach allows for use of either the subtraction or percentage methods, depending on chemical-specific circumstances. The subtraction method is acceptable when other sources of exposure (i.e., other than drinking water and fish exposure) can be considered “background” and can be subtracted from the RfD, thus reducing the amount of the RfD “available” for water-related sources of intake. If adequate data exist to quantify exposure from sources other than the source of concern (i.e., drinking water), the percentage method can be used in which the percentage of total exposure typically accounted for by drinking water (RSC) is applied to the RfD to determine the maximum amount of the RfD “apportioned” to drinking water. With both procedures, a “ceiling” level of 80% and a “floor” level of 20% of the RfD apportioned to drinking water are applied. Use of these limits ensures the total exposure is maintained below the RfD while generally avoiding an extremely low limit in a single medium that represents just a relatively minor fraction of the total exposure.

While the subtraction method is acceptable when only one criterion, standard, or guidance is available for a particular chemical, the percentage method is recommended when multiple media criteria are at issue. The USEPA generally compares the multiple source exposures with one another to estimate their relative contribution to the total.

## 2.1 Data Identification

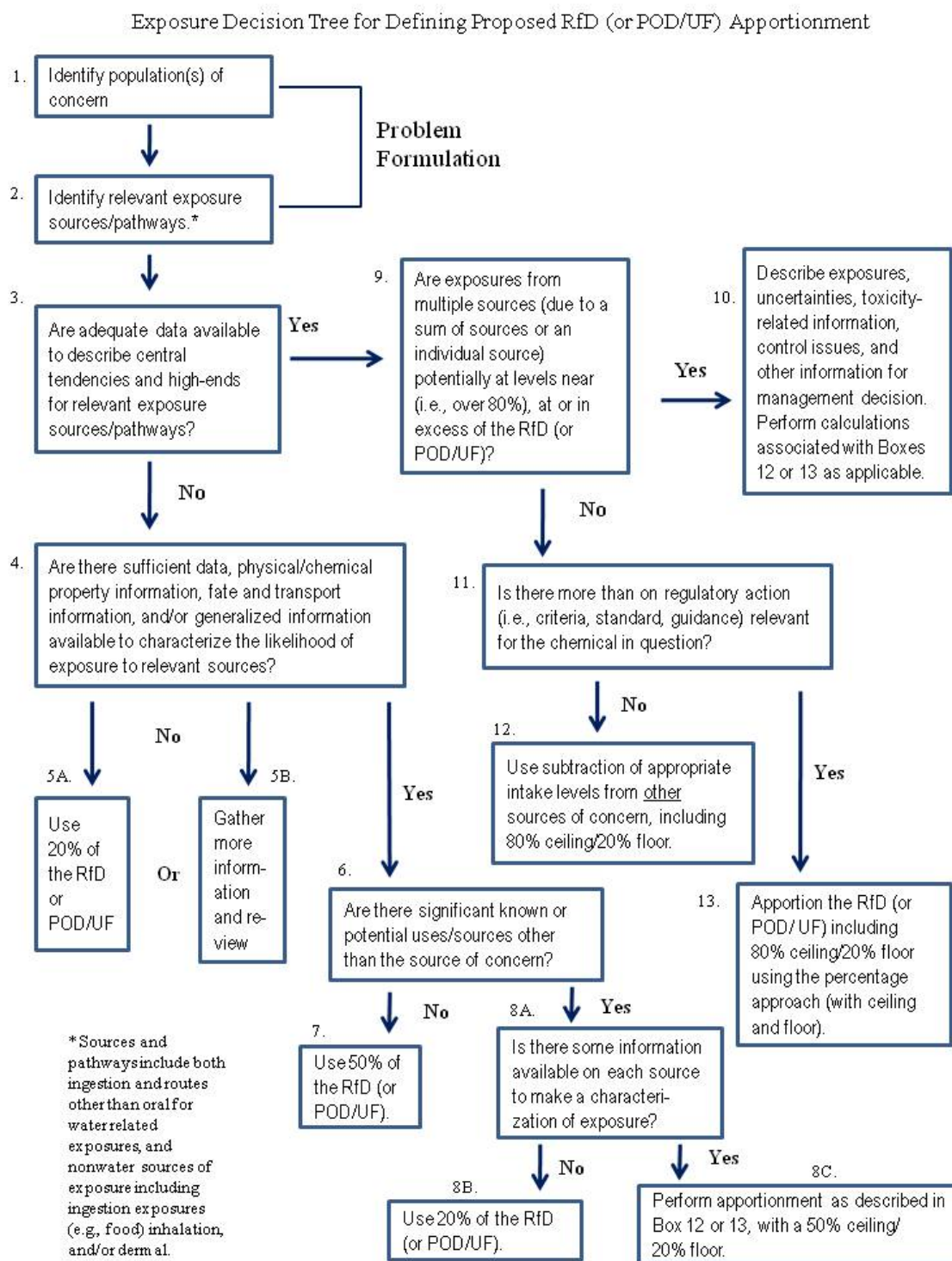
A literature search was initiated and completed in mid-2007 for scientific articles related to RDX. Computerized searches for peer-reviewed literature and technical reports were completed using the following online search services:

- MEDLINE (PubMed)
- Defense Technical Information Center (DTIC<sup>®</sup>)
- SciFinder
- Internet searches using Google

The search terms used included the chemical name RDX and various combinations of *plants*, *consumption*, *bioaccumulation*, *exposure*, *toxicokinetics*, and *pharmacokinetics*. The Google search also included *bioavailability*, *plant uptake factor*, *bioconcentration ratio*, *toxicity*, *animal*, *human*, *oral*, *dermal*, and *inhalation*. An electronic alert system was established in MEDLINE to identify any RDX-related materials published during the course of the project.

In addition, secondary published sources were reviewed, e.g., *Toxicological Profile for RDX* (ATSDR, 1995), as were secondary online sources such as the USEPA Integrated Risk Information System (IRIS) (USEPA, 2008). Furthermore, meetings were held with the US Army, which maintains a large inventory of peer-reviewed published and non-published technical reports on various aspects of RDX.

Non-published reports related to the environmental investigations of DoD sites under the Defense Environmental Restoration Program were not examined as part of the literature search. Unless investigations/studies related to these sites were presented in the peer-reviewed literature, data from these sites were not evaluated for this effort.



**Figure 1** Exposure Decision Tree. Taken from *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health* (USEPA, 2000).

Monitoring of published literature for peer reviewed articles related to RDX is conducted by the US Army Center for Health Promotion and Preventive Medicine (USACHPPM). Articles identified through the Army's monitoring were evaluated as they were provided.

Specific chemical use databases were searched to address potential sources of RDX to the environment including:

- National Library of Medicine (NLM) Hazardous Substance Data Bank (HSDB, 2009)
- National Institute of Health (NIH) Household Products Database (NIH, 2008)
- Pesticide Product Information System (PPIS) (USEPA, 2009a)

## **2.2 Regulation of RDX**

When compared to other chemicals used by the Army, there are few environmental regulations, standards, or guidance values for RDX. International, federal, and state standards and guidelines for RDX are presented in Appendix A. This information was gathered from the Bureau of National Affairs Environmental and Safety Library<sup>1</sup> and internet searches of various government agency web sites. The state regulatory data presented is not comprehensive, as the data search focused primarily on states where Army ammunition plants (AAPs) are located. Thus, additional state standards/guidelines for RDX may exist. In addition, RDX is also regulated in the US as a high production volume chemical under the Toxic Substances Control Act and, internationally, by the Organization for Economic Cooperation and Development.

As discussed in Section 4, human health toxicity benchmarks are available in the USEPA IRIS, but are currently undergoing revision. Although not regulated under the SDWA, RDX is included on the draft Contaminant Candidate List 3 and the Unregulated Contaminant Monitoring Rule 2, indicating the USEPA is evaluating the appropriateness of developing a drinking water standard. The USEPA does have health advisory values for RDX in drinking water and several regional USEPA offices have established risk-based screening levels for use in risk assessments. More importantly, an increasing number of states have moved ahead and developed standards and guidelines for RDX in soil, groundwater, and drinking water.

## **3. Properties and Sources**

RDX is a white crystalline solid; its odor and taste are not described (ATSDR, 1995). As with most chemicals, the fate and transport of RDX within the environment is largely governed by its physical and chemical properties. The sources of RDX are important for determining potential exposure routes for people living near current or former military facilities.

### **3.1 Environmental Fate and Transport Properties**

As noted in Table 1, the vapor pressure of RDX is very low suggesting evaporation of RDX from its solid phase does not contribute to environmental transport. Similarly, RDX has a low Henry's

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<sup>1</sup> <http://www.bna.com/>

Law Constant, resulting in an inconsiderable volatilization from water; thus, volatilization is not considered an environmental fate pathway.

RDX is slightly soluble in water, but it has been detected in both groundwater and surface water at or near AAPs that manufacture the chemical or assemble munitions containing the explosive (Talmage et al., 1999). The logarithm of the octanol/water partition coefficient ( $K_{ow}$ ) for RDX is estimated to be 0.87, indicating RDX is likewise not very lipid soluble and therefore its potential for bioaccumulation in aquatic organisms is relatively low. Limited investigation indicates the RDX bioaccumulation factor (BAF) for aquatic organisms is about 10 (USACHPPM, 2006).

**Table 1** Physical and Chemical Properties of RDX.

Identification	RDX	Reference*
CAS Number	121-82-4	HSDB 2009
Molecular Formula	$C_3H_6N_6O_6$	HSDB 2009
<b>Physical and Chemical Properties</b>		
Boiling Point (°C)	276–280	HSDB 2009
Melting Point (°C)	205.5	HSDB 2009
Molecular weight (g/m)	222.12	HSDB 2009
Log $K_{oc}$	0.84–2.2	USACHPPM 2002a
Log $K_{ow}$	0.87	HSDB 2009
Water Solubility (mg/L at 25 °C)	59.7	HSDB 2009
Vapor Pressure (mm Hg at °C)	$4.1 \times 10^{-9}$	HSDB 2009
Henry's Law Constant (atm-m <sup>3</sup> /mole)	$1.2 \times 10^{-5}$	USACHPPM 2002a

\*HSDB = NLM's Hazardous Substances Data Bank

\*USACHPPM

The range of  $K_{oc}$  values for RDX indicates its medium-to-high mobility in soil (ATSDR, 1995); therefore, RDX can be expected to leach into groundwater. Such mobility is supported by experimental data, which have shown that RDX is not readily bound or retained in soil (Dontsova et al., 2006). Based on these findings, adsorption to sediment and particulate matter in the aquatic environment should not be substantive; however, adsorption would increase with increased organic matter or clay content of the sediment. Plants have been shown to bioaccumulate RDX from contaminated soil and irrigation water and concentrations greater than those in soil and hydroponic media have been reported (Harvey et al., 1991; Checkai and Simini, 1996; Checkai et al., 1996; Price et al., 1997).

RDX hydrolyzes slowly under ambient conditions (Hoffsommer and Rosen, 1973).

Anaerobically, biotransformation of RDX occurs by sequential reduction of the nitro groups to amine groups. These reductions destabilize the ring structure leading to ring cleavage and the formation of hydrazine, formaldehyde, and methanol (McCormick et al., 1981), all of which are subject to more rapid mineralization than RDX (USAMRDC, 1980). RDX in fresh water has been shown to undergo photolysis in natural sunlight (Burton and Turley, 1995). RDX degraded

with a half-life of 0.7 days at the surface of river water in summer and with a half-life of 53 days at a depth of 3 meters (m) in winter (USAMRDC, 1983).

Evaluation of the degradation of RDX in soil has generated somewhat disparate results. A half-life of about 29 days was reported for RDX under well-drained conditions compared with a half-life of about 4 days in soil that was 58% sand, 35% silt, 7% clay (sandy loam), with a pH of 6.7, 10.4% organic matter, and initial cell biomass of  $6 \times 10^{11}$  cells/kilogram (kg) (Ringelberg et al., 2003). The faster decomposition in the saturated soil may have been due to more anaerobic conditions. In soil that was 77% silt, 21% clay, 2% organic carbon, and with a pH of 5.6 (cell biomass not provided), 74% of the RDX remained after 60 days (Harvey et al., 1991). The differences in these outcomes may have been due to the microbial populations in the two experiments because Ringelberg et al. (2003) demonstrated the degradation of RDX was dependent upon microbial activity. Therefore, half-life in soil may be concentration-dependent.

### 3.2 Sources

RDX is a synthetic chemical unknown to occur naturally. RDX is not produced commercially in the US (HSDB, 1994, as cited in ATSDR, 1995). Rather, production is limited to the Holston AAP in Kingsport, Tenn. Several AAPs, such as Louisiana (Shreveport), Lone Star (Texarkana, Tex.), Iowa (Middletown), and Milan (Milan, Tenn.) also handle and package RDX (ATSDR, 1995). Common military uses of RDX have been as an ingredient in plastic bonded explosives, or plastic explosives that have been used as explosive fill in almost all types of munitions compounds.

RDX has been released from Army manufacturing and munitions loading facilities as well as firing ranges (Jenkins et al., 2006). RDX reaches the environment via the waste streams at production facilities, currently limited to the Holston AAP, but also via the waste streams of facilities loading and packaging munitions. In addition, RDX can enter the environment as a result of disposal/demilitarization of munitions. At artillery range impact areas, the major residues of RDX are from the military-grade Composition B, of which RDX is a component, used in artillery and mortar rounds. Its residues are very heterogeneously distributed at artillery range impact areas and can be described as randomly distributed point sources (USACE, 2005). As a result of activities associated with the production, use, and disposal of RDX and the munitions in which it is used, varying amounts of the chemical may be expected to be found in soil, groundwater, surface water, sediment, and biota in areas where it was released into the environment.

The Agency for Toxic Substances and Disease Registry (ATSDR, 1995) has reported that RDX has had civilian applications including use in fireworks, demolition blocks, as a heating fuel for food rations, and occasionally as a rodenticide. The reported use of RDX in fireworks was investigated because such usage could potentially result in widespread dispersion in the environment. The 1995 ATSDR Toxicology Profile for RDX (ATSDR, 1995) is widely cited as the reference for the use of RDX in civilian fireworks. However, the exact reference relied upon by the ATSDR for this claim appears incorrect. The following potential reference sources for the statement that RDX is used in civilian fireworks can be found in that document (ATSDR, 1995):

Page 66 “Civilian applications of RDX include use in fireworks, in demolition blocks, as a heating fuel for food rations (Turley and Brewster, 1987), and as an occasional rodenticide (HSDB, 1994).”

*and*

Page 79 “...although it has been used occasionally as a rat poison or for civilian uses, such as in fireworks or as heating fuel for food rations (Merck, 1989; HSDB, 1994; Turley and Brewster, 1987).”

The sources of the statements made in the ATSDR report were examined and none report the use of RDX in civilian fireworks. The paper by Turley and Brewster (1987) discusses the civilian uses of RDX in the introduction of the paper; however, it fails to produce a reference for the claim that RDX is used in fireworks. The research presented in the paper is unrelated to fireworks or heating fuels. The second source, listed as Merck (1989), reports the use of RDX as a rat poison and as a high explosive, but it does not report its use in civilian fireworks. Finally, the most recent online edition of the HSDB (2009) was searched (the 1994 version is no longer available). The current online HSDB does not report that RDX is used in fireworks. In addition, the National Fireworks Association (NFA) responded to inquiries with a statement that “any reports in the open literature of use of RDX in fireworks must be considered suspect” (NFA, 2008). Thus, the civilian exposure to RDX as any part of commercial fireworks is unlikely.

As mentioned above, the ATSDR (1995) also reported RDX occasionally was used as a rodenticide. Inquiry to the USEPA-sponsored PPIS revealed that RDX was never listed as a pesticide active ingredient in a registered product (USEPA, 2009a). Therefore, the potential for RDX to enter the environment from use as a pesticide is also unlikely.

Crowson et al. (1996) and Cullum et al. (2004) sampled for RDX on police personnel and in public and private areas of several cities in the United Kingdom (UK). The sampling conducted was designed to determine ambient concentrations of explosives in urban environments in support of police forensic investigations. They found that RDX is unlikely to be found in urban environments, supporting the assumption that RDX exposure will be limited to military facilities and operations. See Section 5.5 for further discussion.

Occupational exposure is limited to those workers who handle RDX in its manufacture or use and exposures could include inhalation of dust and dermal contact with the skin. The National Institute of Occupational Safety and Health’s (NIOSH’s) most recent estimate (1981–1983) was that 488 workers were potentially exposed to RDX in the US (NOES, 1990).

## **4. Health Effects**

The health effects of RDX have been reviewed in depth by several different authors (Cholakis et al., 1980; USEPA, 1988, 2008; Etnier et al., 1990; ATSDR, 1995). Studies in experimental animals have evaluated the potential of RDX to cause acute toxicity, systemic toxicity following short- and long-term exposures, as well as carcinogenicity. Significant questions remain regarding the potency of RDX as a carcinogen (Parker et al., 2006).

## 4.1 Non-Cancer

Based on the review of the available studies, the USEPA's IRIS (USEPA, 2008) selected the two-year rat feeding study (US DoD, 1983) for derivation of the RfD. The USEPA determined this was a well-designed and a well-executed study with a large number of dose groups using an adequate number of animals. Based on this study, the USEPA established a No Observed Effect Level of 0.3 mg/kg-day and Lowest Observed Adverse Effect Level of 1.5 mg/kg-day for inflammation of the prostate in rats, resulting in an RfD of 0.003 mg/kg-day after application of uncertainty factors of 100.

## 4.2 Cancer

The USEPA evaluated the available carcinogenicity data and classified RDX as a Group C (possible human carcinogen) using the USEPA (1986) cancer risk assessment guidelines (USEPA, 1988). The classification was based on hepatocellular adenomas and carcinomas in female B6C3F1 mice in the US DoD (1984) study.

A recent study (Parker et al., 2006) reevaluated the archived histological sections from the B6C3F1 mouse study using current histopathological diagnostic criteria and interpretations. Results of the reevaluation indicated a slightly lower incidence, due to the reclassification of hepatocellular adenomas as foci of cytoplasmic alteration, at each of the doses (7.0, 35.0, or 100 mg/kg-day) in female mice. According to these authors, a Pathology Working Group (PWG) reviewed the reevaluation and arrived at a consensus classification of each lesion. Based on the consensus diagnoses of the PWG, Parker et al. (2006) reported only one female group (35 mg/kg-day) showed a significant increase compared to the control group. Since the incidence of hepatocellular neoplasms for all groups—including the 35 mg/kg-day group—was within the reported incidence range for spontaneous hepatocellular neoplasms in female B6C3F1 mice, the authors concluded the incidence observed in the 35 mg/kg-day group can be interpreted as equivocal evidence of carcinogenic effect. USEPA (2009b) indicates that the carcinogenicity of RDX is being reevaluated.

## 4.3 Absorption and Bioavailability

No studies were identified that estimated the extent of absorption of RDX following oral, inhalation, or dermal route of exposure in humans. However, ATSDR (1995) reviewed several studies that observed toxic effects (including neurotoxicity) following oral and inhalation exposures, indicating that RDX can be absorbed through the gastrointestinal (GI) system or into the lungs (Kaplan et al., 1965; Merrill, 1968; Hollander and Colbach, 1969; Stone et al., 1969; Ketel and Hughes, 1972). In another study, 90% of the RDX was no longer detected on the skin after one hour, and none was detected after 48 hours (Twibell et al., 1984; as cited in ATSDR, 1995).

Acute oral lethality studies and single dose experimental studies with RDX in experimental animals have also indicated RDX is absorbed through the GI tract (Schneider et al., 1977, 1978). In a toxicokinetic study, the bulk of the RDX was transported across the GI absorption barrier as evidenced by less than 3% of RDX being recovered in the feces when 100 mg/kg RDX/kg was



administered to rats by gavage (Schneider et al., 1977). Administration of 50 mg/kg  $^{14}\text{C}$  RDX to the rats resulted in most of the radioactivity being found in the liver and urine after 24 hours, with further partitioning to other parts of the body during the next three days. Overall, 43% of the radioactivity was expired as  $^{14}\text{CO}_2$ .

Crouse et al. (2008) investigated the potential of soil-bound RDX to be absorbed through the GI tract of rats. Results from this study indicate the adsorption of RDX to soil particles rendered it less efficiently absorbed and likely to be less bioavailable for uptake, and that bioavailability varied with soil type.

Although skin contact with RDX-contaminated soil can represent an important route of exposure to RDX, no absorption data following dermal exposure in humans *in vivo* were identified. Several percutaneous absorption studies using human, pig, and animal skin *in vitro* (Reddy et al., 2008; Reifenrath et al., 2002, 2008) were identified that estimated dermal absorption ranging from 0.04–2.6% of the applied dose depending on the carbon content of the soil, soil type, and age of the soil.

## 5. Occurrence

Early waste disposal practices in manufacturing, as well as incomplete detonations at ranges, have led to various levels of contamination of soil, surface water, and groundwater at military facilities in the US by energetic residues, including RDX (Jenkins et al., 2005; Pennington et al., 2005; Clausen et al., 2007). Only 24 sites on the USEPA's National Priorities List (NPL) have detected RDX and all of these sites are associated with munitions-related activities.

The occurrence of RDX at Superfund and DoD environmental investigation sites was investigated to determine the potential for these sites to act as source areas given their intended civilian use. All identified environmental restoration sites contaminated with RDX are associated with DoD activities. The 24 sites listed in Table 2 are from the USEPA Superfund Site Information Database.<sup>2</sup> The Superfund Site Information Database contains information on hazardous waste sites, potentially hazardous waste sites, and remedial activities across the nation, including sites that are on the NPL or being considered for the NPL. Each of the 24 listed sites is associated with current or former DoD activities involving the use of munitions or munition-related items. The DoD reports there are 76 active military sites with RDX contamination, nine closed sites (closed under one of the multiple Base Realignment and Closure Acts), and 15 sites under the Formerly Used Defense Sites (FUDS) program.<sup>3</sup> Given that all sites under the FUDS program have not been sampled, it is likely that additional sites within this category could be identified. At each of the Superfund and DoD sites where RDX was identified as a contaminant, it is not necessarily the chemical driving a response action nor is it likely to be the only chemical contaminant.

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<sup>2</sup> See the USEPA Comprehensive Environmental Response, Compensation, and Liability Information System (CERCLIS) at <http://www.epa.gov/superfund/sites/cursites/>. Searched December 2008.

<sup>3</sup> Office of the Deputy Under Secretary of Defense for Installations and Environment, December 2007.

**Table 2** USEPA Superfund Site Information Database, Sites with Reported Detections of RDX.

EPA ID	Site Name	City	State	NPL Status
AL3210020027	Anniston Army Depot (Southeast Industrial Area)	Anniston	Ala.	Final
WA5170027291	Bangor Naval Submarine Base	Silverdale	Wash.	Final
WA7170027265	Bangor Ordnance Disposal	Bremerton	Wash.	Final
CA3570024551	Castle Air Force Base (six Areas)	Merced	Calif.	Final
NCD095459392	Chemtronics, Inc.	Swannanoa	N.C.	Final
NE2213820234	Cornhusker AAP	Grand Island	Neb.	Final
CA7210020676	Fort Ord	Marina	Calif.	Final
ID4890008952	Idaho National Engineering Laboratory	Idaho Falls	Idaho	Final
IA7213820445	Iowa AAP	Middletown	Iowa	Final
IL0210090049	Joliet AAP (Load-Assembly-Packing Area)	Joliet	Ill.	Final
IL7213820460	Joliet AAP (Manufacturing Area)	Joliet	Ill.	Final
CA2890090002	Lawrence Livermore National Lab (Site 300)	Tracy	Calif.	Final
LA0213820533	Louisiana AAP	Doyline	La.	Final
TN0210020582	Milan AAP	Milan	Tenn.	Final
VA7170024684	Naval Surface Warfare Center – Dahlgren	Dahlgren	Va.	Final
VA8170024170	Naval Weapons Station – Yorktown	Yorktown	Va.	Final
NE6211890011	Nebraska Ordnance Plant (Former)	Mead	Neb.	Final
MA2570024487	Otis Air National Guard Base/Camp Edwards (Mass. Military Reservation)	Falmouth	Mass.	Final
WA4170090001	Port Hadlock Detachment	Indian Island	Wash.	Deleted
IL8143609487	Sangamo Electric Dump/Crab Orchard National Wildlife Refuge	Cartersville	Ill.	Final
IL3210020803	Savanna Army Depot Activity	Savanna	Ill.	Final
NY0213820830	Seneca Army Depot	Romulus	N.Y.	Final
OR6213820917	Umatilla Army Depot (Lagoons)	Hermiston	Ore.	Final
MD0170023444	US Naval Surface Warfare Center – White Oak	Silver Spring	Md.	Not Listed

A review of published literature reporting environmental measurements of RDX in the US identified 34 military sites with RDX measurements in surface water, groundwater, soil, and sediment. Appendix B summarizes the available environmental measurements data from these published reports. Concentrations at these sites in the various media range widely. Reported concentrations of RDX ranged from less than detection limits to 13,900 mg/kg in soil, 0.004–110 milligrams per liter (mg/L) in surface water, >0.001–36 mg/L in groundwater, and 2–120,000 mg/kg in sediment. However, this finding is suspect because the reported RDX concentration in surface water (110 mg/L) is higher than the water solubility of RDX.

The low water solubility of RDX, the slow dissolution in aqueous solution, low vapor pressure, and a low affinity for hydrophobic substances would predict RDX would have limited retention in soil. However, its potential to leach from soil has been demonstrated by laboratory and field studies. RDX is reported to pass through laboratory soil columns with minimal retardation and reduction (McGrath, 1995). Pennington et al. (1995) reported that RDX readily leached from clay loams and soils collected from several different sites. In laboratory investigations using a

variety of soils ranging from clay to sandy loam, Cataldo et al. (1990) reported that less than 2% of RDX was bound as a non-extractable residue.

Although the relatively slow dissolution of RDX may limit its migration to groundwater, RDX is persistent and relatively mobile once it dissolves. The propensity to be relatively mobile through soil favors its transport from soil to groundwater as well as the potential to migrate offsite to groundwater or other water sources. The potential to migrate from soil to water has been supported by few onsite and offsite groundwater and surface water studies that reported energetic compounds, including RDX, from impact areas and at firing points (see Appendix B).

No human intake data were located for RDX in water, food, etc. The following sections summarize the available environmental measurement data located, primarily from military facilities in the US.

### **5.1 Drinking Water (Groundwater and Surface Water)**

Data on concentrations of RDX in drinking water are not available. RDX is not a regulated contaminant under the SDWA and no data on RDX occurrence in drinking water are available. However, the USEPA has included RDX as a contaminant to be monitored in the second five-year cycle (2007–2011) under the Unregulated Contaminant Monitoring Regulation (UCMR) program (USEPA, 2005). These data will not be available until 2011.

### **5.2 Surface Water**

Data from Army installation sites were located that detected energetic residues including RDX in various media including surface water (see Appendix B). Of the 34 installation sites investigated in the available reports, surface water bodies are found on only five sites. Two of these had no detectable RDX concentrations, while the other three had concentrations of RDX that ranged from >0.004–109 mg/L (Small and Rosenblatt, 1974; Envirodyne Engineers, Inc., 1980; Spanggord et al., 1981); the maximum concentration reported is higher than the solubility of RDX in water.

### **5.3 Groundwater**

RDX was detected in groundwater at 11 of the 34 sites at concentrations ranging from 0.00025 mg/L–70 mg/L. Two other sites had no detectable RDX measured in the groundwater. For example, at Cornhusker AAP, onsite groundwater (downgradient of contamination) with an RDX concentration of 0.3 mg/L (not specified whether concentrations were single, average, or maximum values) was reported (Spalding and Fulton, 1988, cited in Talmage et al., 1999). Offsite plumes had RDX concentrations up to 0.1 mg/L (Spalding and Fulton, 1988, cited in Talmage et al., 1999), indicating the potential of RDX to migrate offsite.

### **5.4 Air**

RDX may enter the air through the release of contaminated particulate matter formed during the incineration of RDX-containing mixtures or through evaporation from aquatic effluent streams

or waste storage lagoons (US Army, 1984). However, no data were located regarding measurements of RDX in ambient or occupational air. Indoor samples collected at Holston AAP in 1974 found RDX concentrations ranging from not detected ( $<0.5$  mg/cubic meter [ $\text{m}^3$ ] [ $4.5\text{--}60$  mg/ $\text{m}^3$ ] [546 parts per million (ppm)]) (US Army 1975, cited in ATSDR). In addition, RDX was detected at a concentration of  $0.032$  mg/ $\text{m}^3$  (0.29 ppm) in the particulate fraction of one indoor sample taken from the incorporation area of Holston AAP in 1986 (Bishop et al., 1988, as reported in ATSDR, 1995).

## **5.5 Soil and Dust**

RDX soil concentrations measurements ranged from  $0.01\text{--}13,900$  mg/kg in 24 of the 34 Army installation sites (see Appendix B) for which data were available. Two surveys have been carried out to determine the background concentrations of traces of explosives in public places. One survey concentrated on a variety of areas including transport areas and police stations in and around London, UK, in 1994–1995 (Crowson et al., 1996). In the Crowson et al. study, RDX was detected in eight of 592 samples at amounts ranging from 4–11 nanograms (ng). The total number of samples included clothing samples from 48 police personnel but no RDX was detected on any of the clothing sampled. The second survey (Cullum et al., 2004) examined concentrations in four major cities (Birmingham, Manchester, Cardiff, and Glasgow) in the UK and samples were taken (by wiping a surface with a swab or by air vacuum onto a filter paper) from transport sites, hotel rooms, private houses, taxis, buses, trains, airports, privately owned vehicles, and clothing purchased from charity shops. Only one of 422 samples had detectable RDX of 7.5 ng. Results of these surveys indicate that RDX is unlikely to be found in urban environments and supports the assumption that RDX exposure will be limited to military facilities and operations.

## **5.6 Sediment**

RDX is not sampled as part of any identified national or regional sediment sampling program. However, RDX concentrations up to  $120,000$  mg/kg have been detected in sediments from five of the 34 Army installation sites for which data were available (see Appendix B).

## **5.7 Foods**

RDX is not an analyte that has been measured in the market basket foods surveys conducted by the US Food and Drug Administration (US FDA) Total Diet Study Market Baskets 1991/1993 through 2003/2004 (US FDA, 2004) and no RDX concentration data in food were located in the literature.

Food crops may be grown on land previously used as military training grounds and potentially contaminated with RDX. In addition to being grown or raised on potentially contaminated soil, those crops and animals may be watered with contaminated water. The available data on uptake and bioconcentration/bioaccumulation in plants and animals are discussed in the next two sections.

### 5.7.1 Plants

A survey of a limited number of vegetables obtained from home gardens irrigated with RDX-contaminated well water (within a plume adjacent to an RDX-contaminated munitions site) indicated that the vegetables had no detectable levels of RDX in edible plant tissues (ENSR Consulting and Engineering, 1991).

The potential of RDX to be found in soils and groundwater at sites of munitions production, testing, or disposal raises concerns over the potential bioavailability from soils, surface, and groundwaters from sites contaminated with RDX. Several studies have indicated that RDX is rapidly assimilated by the roots of higher plants (trees) and by forage and crop plants from soils (Cataldo et al., 1990; Fellows et al., 1995; Checkai and Simini, 1996; Checkai et al., 1996; Price et al., 1997) and nutrient solutions (Cataldo et al., 1990; Harvey et al., 1991; Price et al., 1997). Following uptake into the roots, RDX is translocated and partitioned into tissues such as stems, leaves, flowers, and fruit.

Several studies (Cataldo et al., 1990; Harvey et al., 1991; Fellows et al., 1995; Checkai and Simini, 1996; Checkai et al., 1996; Price et al., 1997; Larson et al., 1999) describe different experiments in which a number of garden crops (bush bean, tomato, lettuce, and radish varieties) and field crops (corn, soybean, and alfalfa) were grown in RDX-contaminated soil (Cataldo et al., 1990; Fellows et al., 1995; Checkai and Simini 1996; Checkai et al., 1996; Price et al., 1997; Lachance et al., 2003), in soil irrigated with RDX-contaminated water (Price et al., 1997; Larson et al., 1999), or in RDX-containing water in hydroponic studies (Cataldo et al., 1990; Harvey et al., 1991; Checkai and Simini, 1996; Checkai et al., 1996). In these studies, different plant tissues including edible portions (root, stem, leaf, seed, and fruit) were sampled for RDX and plant uptake factors (PUFs) for RDX were calculated. These studies demonstrate that RDX is readily absorbed by plants from both soil and irrigated water. Plants grown in RDX-contaminated soil bioaccumulated RDX, with plant concentrations greater than soil concentrations. In hydroponic studies, RDX did not appreciably bioaccumulate (Checkai and Simini, 1996), and uptake was not significant in tomato fruit, bush bean seeds, radishes, or soybean seeds. Additionally, plant concentrations in hydroponic studies were generally less than that of the irrigation water.

In a recent study, Fellows et al. (2006) planted corn and alfalfa seeds in soils amended with U-<sup>14</sup>C-labeled and unlabeled RDX at a final RDX concentration of 15 mg/kg. Plants were grown to maturity. Randomly selected plants were harvested and RDX distribution and metabolism were analyzed. Concentrations of non-metabolized parent RDX in corn leaves and alfalfa shoots (stems and leaves) were reported as 19 mg/kg and 187 mg/kg, respectively. Although this study did not estimate PUFs, results indicate the potential of plants to take up RDX from the soil.

Appendix C summarizes results of these studies for species of field and garden crop plants that are known human foods and presents soil-to-plant uptake factors that range from 0.06–5.99 and water-to-plant uptake factors ranging from 0.16–5.50. A similar compilation of uptake factors (or bioconcentration ratios) has been reported in a recent analysis by McKone and Maddalena (2007). Uptake of RDX has also been evaluated in non-edible tissues of both food and non-food crops (e.g., corn [stover, ear, tassel, leaves, or roots], Japanese millet, perennial ryegrass, carrot shoot, spinach seeds, etc.) (Cataldo et al., 1990; Checkai and Simini, 1996; Price et al., 1997; Fellows et al., 1995; Larson et al., 1999; Lachance et al., 2003). Although uptake into these non-

edible portions are generally similar to that reported for the edible portion, uptake factors estimated from these non-edible portions have not been considered in this report.

### **5.7.2 Meat and Fish**

A few studies were located that indicate wildlife are unlikely to take up RDX (see Appendix D). Samples of liver and muscle of white-tailed deer collected at a site used in the past for munition production, storage, and demilitarization and from an offsite location did not contain RDX or its breakdown products in the deer tissues (USACHPPM, 1994). Similarly, no RDX was found in deer and/or white-footed mice sampled from other RDX-contaminated sites (USACHPPM, 1994, 2002). The lack of RDX contamination in these wildlife tissues suggests RDX is not absorbed to an appreciable extent and that wildlife species inhabiting these areas are not likely to bioaccumulate RDX in their tissues.

Fellows et al. (2006) also assessed the potential bioavailability of plant-incorporated  $^{14}\text{C}$ -RDX and plant-derived  $^{14}\text{C}$ -RDX metabolites by a representative hindgut herbivore, the prairie vole. These authors concluded that at the very least, RDX and/or RDX metabolites are capable of uptake and retention by herbivores feeding on plant material growing on RDX-contaminated soil, and that edible tissues containing  $^{14}\text{C}$ -RDX-derived materials are available to subsequent predators, indicating the potential for transfer to a higher trophic level.

RDX is metabolized rapidly and about 30–40% of administered dose is expired as  $^{14}\text{CO}_2$  with some detectable levels in tissues after 24 hours (Schneider et al., 1977, 1978). A physiologically based pharmacokinetic model for RDX in the rat simulates the available kinetic data following intravenous and oral routes and indicates metabolism of RDX is a first order process (Krishnan et al., in press). According to these authors, the model can serve as a basis to investigate the potential pharmacokinetic basis of species differences in toxicity and carcinogenicity of RDX.

A few studies have evaluated the bioaccumulation of RDX in the edible portions (muscle) and viscera of fish including bluegill sunfish, channel catfish, and fathead minnows (Belden et al., 2005; Burrows et al., 1989; Bentley et al., 1977; Layton et al., 1987; Liu et al., 1983) (see Appendix D). Results from these studies indicate fish may contain RDX, with BAFs in the edible portions ranging from 1.5–5.9 mL/g.

## **6. Potential for Human Exposure**

### **6.1 Conceptual Exposure Model**

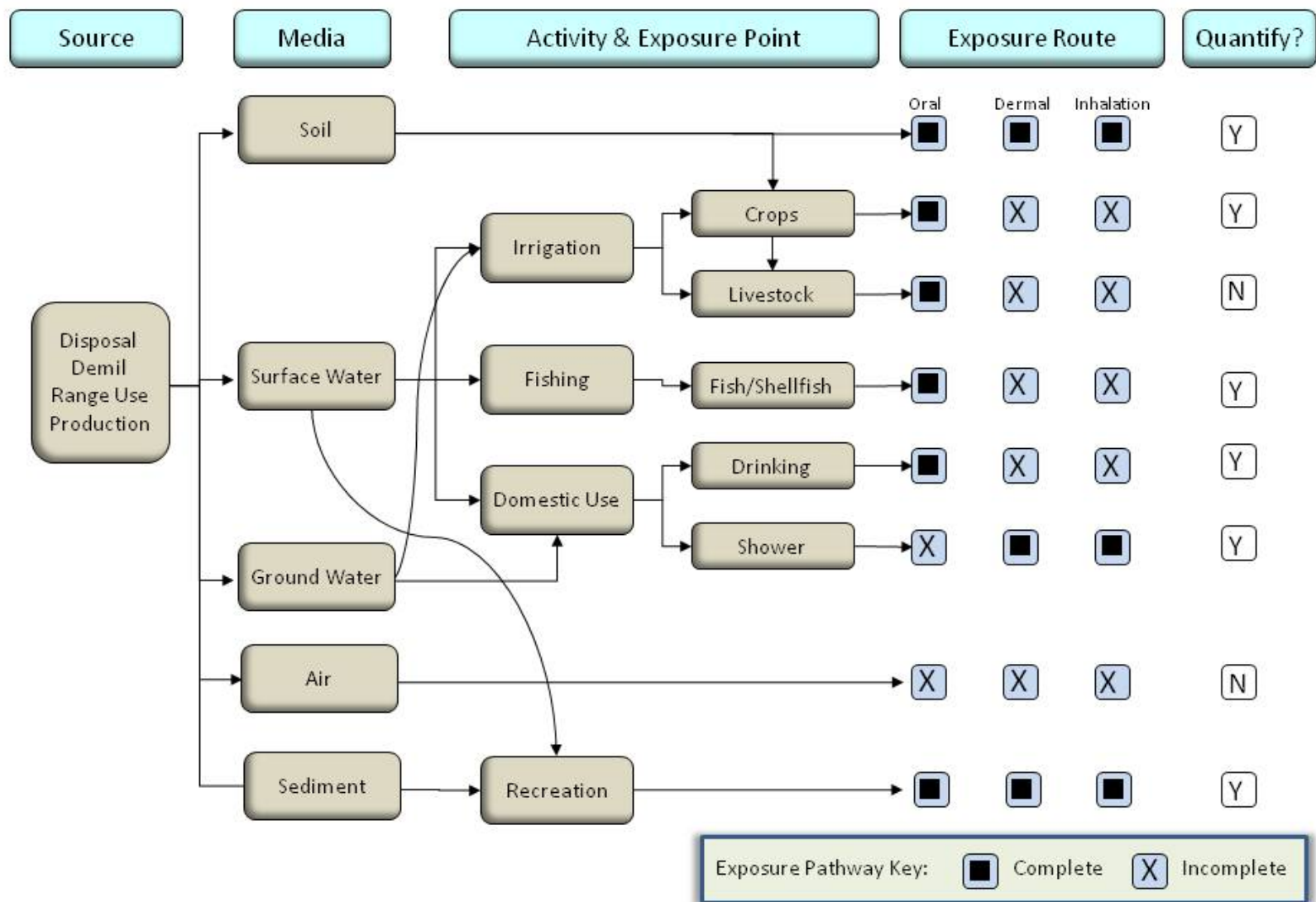
A conceptual exposure model describes the potential pathways of human exposure to RDX. Potential sources of environmental releases, relevant environmental media (e.g., soil, water, sediment), exposure points or activities that may allow RDX to enter the human body, and the exposure routes, were identified. Populations of concern in the general population include those who live on or near a contaminated facility. In addition, if drinking water sources are contaminated or foods grown on contaminated soils are widely distributed, people farther from the source of contamination may be exposed. Using the data presented in the previous sections, the conceptual exposure model in Figure 2 was developed.

### *Environmental Media and Pathways*

Environmental media that may be contaminated to differing degrees include ground- and surface waters, soil, and sediments. The most likely source of general population exposure is ingestion of contaminated drinking water. In addition, ingestion of food (plants and animals) grown on contaminated soil or irrigated with contaminated water, or fish from contaminated bodies of water, should be considered. Dermal contact with contaminated water, sediments, or soils may occur and soil may be incidentally ingested, but these pathways would not be expected to be significant sources of exposure. Inhalation of contaminated particulate matter, including soil or dust particles or those produced during incineration of RDX-containing wastes, may also be a potential pathway, but no data for this pathway were available and this pathway is considered insignificant. Appendix E contains a list of possible human exposure pathways.

### *Complete Exposure Pathways*

Exposure pathways describe the way a substance moves through the environment and reaches or comes in contact with people. Exposure pathways are considered complete when there is a source of the contaminant present, a mechanism to transport the substance from the source to an environmental media (air, water, soil, sediment), a point or activity where people come in contact with the substance, and the substance can enter the body. If any of these steps is missing, the pathway is considered incomplete and would result in no exposure (see Appendix E). For RDX, there are several complete pathways by which people may be exposed, as illustrated in Figure 2.



**Figure 2** RDX Conceptual Exposure Model.



Table 3 lists the potential pathways for RDX exposure for humans. Several of these pathways (e.g., sediment exposure from recreational activities and inhalation of particulates from incineration) are not significant or quantifiable because they represent infrequent activities with only occasional exposure. While some measurements of RDX in sediments have been made on current or former military facilities, the available data do not support exposure to contaminated sediments offsite. Incidental oral ingestion of water during showering is a relevant pathway, but it is not considered further because the assumption of daily drinking water consumption takes into account all water consumed during the day; therefore, this pathway is not quantified.

**Table 3** Potential Exposure Pathways for RDX.

Environmental Media	Potential Exposure Pathways
Soil	<ul style="list-style-type: none"> <li>• Direct contact with skin</li> <li>• Incidental ingestion of soil</li> <li>• Inhalation of soil particles*</li> <li>• Ingestion of food plants and livestock* grown with contaminated soils</li> </ul>
Surface Water	<ul style="list-style-type: none"> <li>• Direct contact with skin</li> <li>• Ingestion of water</li> <li>• Inhalation through showering*</li> <li>• Dermal contact through bathing</li> <li>• Ingestion of fish from contaminated waters</li> <li>• Ingestion of food plants and livestock* irrigated with contaminated water</li> <li>• Ingestion of water from showering*</li> </ul>
Groundwater	<ul style="list-style-type: none"> <li>• Direct contact with skin</li> <li>• Ingestion of water</li> <li>• Inhalation through showering*</li> <li>• Dermal contact through bathing</li> <li>• Ingestion of food plants and livestock* irrigated with contaminated water</li> <li>• Ingestion of water from showering*</li> </ul>
Air	<ul style="list-style-type: none"> <li>• Inhalation of particulates*</li> </ul>
Sediment	<ul style="list-style-type: none"> <li>• Direct contact with skin during recreational activities*</li> <li>• Incidental ingestion of sediment during recreational activities*</li> <li>• Inhalation of particles during recreational activities*</li> </ul>

\*Pathway is a minor source or otherwise not considered in quantification. See text for explanation.

Inhalation of RDX from showering is also not carried forward. The low solubility of RDX in water, combined with its low Henry's Law Constant of 0.87 (see Table 1), indicate that little RDX will volatilize from water. While aerosols containing RDX may be formed, no data were identified regarding the generation of such aerosols.

Ingestion of livestock or animal foods (e.g., deer) is a pathway that is incomplete. Studies have shown that food animals are unlikely to contain RDX in tissues and, therefore, there is no potential for human exposure (USACHPPM, 1994).

### *Available Exposure Data*

Section 5 describes the available environmental measurement data for RDX. RDX has not been systematically surveyed in US ground- or surface waters, drinking water supplies, the food supply, or air. Considerable data exist in the open literature on measured concentrations of RDX in soil, sediment, groundwater, and surface water from US military facilities that produced or used RDX materials. Uptake studies are available on several food plants, as are measurements of RDX in wild animals grazing on contaminated lands and fish bioaccumulation measures. Limited surveys of public spaces in the UK found only a single small detection (7.5 ng) of RDX. RDX is part of the current UCMR sampling of drinking water and final monitoring results will be available in 2011.<sup>4</sup> However, because RDX is almost exclusively used in military applications on military facilities, one would not expect to see detections of RDX in drinking water supplies that are not proximate with a military facility.

## **6.2 Quantification of RDX in Complete Exposure Pathways**

To quantify exposure from the various complete exposure pathways, concentrations were measured from soil, water, and foods. The US Department of Energy Office of Environmental Management developed the Risk Assessment Information System (RAIS)<sup>5</sup> to readily estimate human intakes at contaminated sites. Estimates of chronic daily intakes (CDIs) can be calculated with the RAIS tools. The equations, parameter values, and assumptions used are from the USEPA's Risk Assessment Guidance for Superfund (USEPA, 1989, 1991). The RAIS modeling tool allows users to select specific chemicals, land uses, exposure combinations, parameters, and concentrations to estimate site-specific intakes and risks. Appendix F includes spreadsheets with equations and default parameter values for the residential scenario that can be used to estimate RDX intake to contaminated water and soil, in addition to equations that estimate intakes from agricultural land use and contaminated food. For each potential exposure source (soil, water, or food), a series of equations are used that represent potential routes and pathways of exposure. Appendix F includes a print out of a Microsoft Excel file with three tabs—one each for water, soil, and food. Within each tab are the equations to represent the potential pathways of exposure for that media.

### **6.2.1 Uptake of RDX by Plants and Bioaccumulation in Fish**

To determine intake of RDX from food, site-specific sampling of food would be desirable. However, if there is limited information on site-specific uptake of RDX from soil into edible portions of garden plants or field crops, plant uptake can be calculated using experimentally-derived PUFs (i.e., soil-to-plant concentrations factor). Similarly, if fish tissue concentrations are not available, uptake of RDX from water by fish can be estimated by multiplying the water concentration by a BAF.

To estimate the chemical concentrations in the edible portions of produce, the USEPA-recommended equation (USEPA, 2005) to determine soil screening levels (SSL) for the soil-plant-human exposure pathway is used. This equation is appropriate for both belowground and

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<sup>4</sup> Preliminary data from the UCMR program is available at: <http://www.epa.gov/safewater/ucmr/data.html#UCMR2>

<sup>5</sup> <http://rais.ornl.gov/>

aboveground vegetation, provided the appropriate PUF is used. The SSL equation for the soil-plant-human pathway is given by:

$$C_{\text{plant}} = C_s / \text{PUF}$$

where:

$C_{\text{plant}}$	= Contaminant concentration in dry plant tissue ( $\text{mg/kg}^{-1}$ )
$C_s$	= Contaminant concentration in soil ( $\text{mg/kg}^{-1}$ dry weight)
PUF	= Soil-to-plant concentration factor

SSLs based on concentrations in plants and the PUF require the use of dry masses of plant and soil. If  $C_{\text{plant}}$  is reported as wet weight, a dry weight-wet weight conversion factor must be applied.

In the plant uptake studies for RDX, experimentally-derived PUF values for edible portions of representative fruits and vegetables ranged from 0.06–79 for food crops grown on RDX-contaminated soil (see Appendix C). Since the range covers several orders of magnitude (more than 1,000), use of the geometric mean (1.60) is appropriate if a central tendency is preferred. PUF values ranged from 0.07–5.5 for food crops irrigated with RDX-contaminated water, with a geometric mean of 0.44.

Uptake of RDX from water by fish can be estimated by multiplying the water concentration by a BAF. Using the available data on RDX measurements in fish (see Appendix E), the experimentally-derived BAF values ranged from 1.7–5.9 mL/g for the limited number of fish species for which data were located (see Appendix D for study details), the geometric mean is 3.3 mL/g (L/dg) and 5.0 mL/g (L/kg) represents the 90<sup>th</sup> percentile.

### 6.2.2 Exposure Parameters

The USEPA provides default exposure parameters to use for site assessments (USEPA, 1989, 1991). Appendix F includes the CDI equations for each pathway (e.g., ingestion of water pathway), along with a table of default parameter values based on USEPA guidance. Some variables (e.g., averaging time and body weight) remain constant among the CDI calculations, but others (e.g., ingestion rate) vary depending on the exposure pathway and if the receptor is a child or adult. A complete list of the default parameter values and references can be found in Appendix F and is available on the RAIS website.

Water, soil, and plant concentrations are inputs to the intake equations. Site-specific measured values are preferred. However, for fish and plant intakes, fish BAFs or PUFs can be used to calculate intake. The calculation table is set up using standard default parameters so that CDIs can be calculated for children, adults, or a person who is exposed through both child- and adulthood. The water, soil, and plant concentration inputs as well as individual parameter values can be changed if situation-specific information is available.

## 7. RSC Calculation

The RSC for RDX is derived by application of the Exposure Decision Tree approach published in USEPA's *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health* (USEPA, 2000). The purpose of the RSC is to account for identified sources and routes of non-occupational exposures to a particular chemical and to apportion allowable amounts from each source so that an individual would not have a total (aggregate) exposure greater than the RfD. RSCs are calculated for chemicals that are non-carcinogens or non-threshold carcinogens. Exposures to RDX are expected to be local and limited. Populations near current or former military installations may be exposed to RDX that has migrated from the site. The primary route of potential exposure of RDX released into the environment is expected to be the ingestion of contaminated surface and groundwaters that have migrated offsite. The Decision Tree process (see Figure 1) provides a framework to evaluate the adequacy of the available exposure data, exposure scenarios, and relevant sources of exposure.

*Problem Formulation* is the first step of the decision framework and includes the first two boxes as:

***Box 1 – Identify populations of concern***

As discussed in Section 6, exposure to RDX is not anticipated to be a national exposure concern; rather there may be exposures to local populations from military facilities that produce or use/d RDX.

***Box 2 – Identify relevant exposure sources/pathways***

The conceptual exposure model, as discussed in Section 6, identified the relevant potential exposure sources to be soil, groundwater, and surface water. Potential pathways include ingestion of soil, water, and contaminated local crops and fish, and dermal contact with soil and from water used in bathing.

Intake assumptions for each route of exposure and exposure parameters can be based on those used in the USEPA Superfund program and found in Appendix F. CDI estimates for a child (6 years), an adult (24 years), and combined child/adult (30 years) may be estimated.

*Data Adequacy*

***Box 3 – Are adequate data available to describe central tendencies and high-ends for relevant exposure sources/pathways?***

Distributional exposure concentration data are not available for RDX; therefore, move to Box 4.

***Box 4 – Are there sufficient data, physical/chemical property information, fate and transport information, and/or generalized information available to characterize the likelihood of exposure to relevant sources?***

RDX was measured in soil, sediment, and ground- and surface waters at several current and former military facilities. The likely exposure to RDX by the general population will be as a result of its manufacture or use at military facilities; therefore, RDX exposure if it

happens would be a localized problem. It is possible for RDX to move offsite and contaminate offsite areas. Current or former military-related sites will be remediated and converted to civilian uses, thereby exposing resident populations to any residual contamination. As described in Section 3, the physical/chemical properties of RDX—the low water solubility of RDX, the slow dissolution in aqueous solution, low vapor pressure, and a low affinity for hydrophobic substances—would predict that RDX would have limited retention in soil. However, its potential to leach from soil has been demonstrated by laboratory and field studies. Despite its slow dissolution that may limit its migration to groundwater, RDX is persistent and relatively mobile once it dissolves, a feature that favors its potential to migrate offsite to groundwater or other water sources. The low volatility of RDX suggests that RDX is not likely to be found in significant concentrations in air and inhalation exposure of particulates from explosions is not considered significant. There are no data on RDX concentrations in the national food supply, but RDX is taken up by plants, therefore food grown on contaminated soil or watered with contaminated water is a possible exposure source.

Given the physical/chemical properties and the available data on RDX at current or former military sites, there are adequate data to determine populations might be exposed to RDX from several exposure sources; therefore, move to **Box 6**.

***Box 6 – Are there significant known or potential uses/sources other than the source of concern?***

The primary use of RDX is in military operations. Civilian blasting uses of RDX are very limited and would not be expected to result in significant exposures to the general population as evidenced by the lack of RDX in public places (Crowson et al., 1996; Cullum et al., 2004), the lack of RDX in commercial household products (HSDB, 2009), and the lack of RDX in commercial pesticides (USEPA, 2009a). There are no data to indicate the US population would be exposed to RDX from any environmental media on a national basis. RDX is not expected to be in the national food supply as there is not widespread RDX contamination of agricultural lands, nor would it be expected to be found in air based on its physical properties. RDX would not be expected to be found to any significant extent in the nation's drinking water, but it is currently on the UCRM monitoring list, which should confirm this assumption. Therefore, on a national basis, there are no other potential sources. Move to **Box 7 and the default RSC of 50%**.

However, local populations may be exposed to RDX as a result of military uses. It is reasonable that both water and soil may be contaminated and area residents may consume foods grown on contaminated soil or irrigated with contaminated water. They may also eat fish from contaminated local waters. A site or location-specific RSC might be warranted. To calculate this RSC, one would use the measured concentrations in soil, water, and foods. If food plant measurements are not available, one could use PUFs from the literature to estimate plant concentrations and corresponding intakes. If no fish sampling is available from the local waterways, one could calculate intake of RDX from fish using the water concentration and a BAF. Using these values, one can estimate intake from various media sources from appropriate exposure scenarios and pathways with

algorithms such as those used by the USEPA Superfund program and described in Appendix F.

***Box 8A – Is there some information available on each source to make a characterization of exposure?***

Yes. See **Box 6** above for local scenario if adequate data are available.

***Box 8C – Perform apportionment as described in Box 12 or 13, with a 50% ceiling/20 % floor).***

While there are no regulatory actions relevant to RDX at the current time, it may be found in soil and water at current and former military sites; therefore, the percentage apportionment approach as described in **Box 13** would be followed.

## **8. Conclusion**

Available data indicate exposure to RDX is not widespread in the environment given its almost exclusive military use. Although RDX concentrations in soil, sediment, groundwater, and surface water are available from a number of military installations, concentrations in the various media range widely, reflecting the local usage patterns. Although some sites have soil, groundwater, and/or surface water measurements, the data are not amenable to developing a meaningful national upper bound or central tendency estimates.

RDX is not a chemical regularly monitored in drinking water. However, the USEPA plans to evaluate the potential for national level exposure using data collected under the UCMR, although data for RDX will not be available until 2011. There are no data on RDX in market basket foods and no consumer products were identified containing RDX. Although it is possible farmers may grow crops in RDX-contaminated soils or irrigate such crops with RDX-contaminated water at these military bases, the soils and water are expected to be remediated to “safe” levels prior to release of the bases for agricultural use. It is, therefore, necessary to ensure that the exposed individual would not have a total (aggregate) exposure greater than the RfD. This is accomplished by developing an RSC for RDX based on estimates of CDI of RDX from all possible sources including soil, water, air, consumer products, and food. Given that available survey data (Crowson et al., 1996; Cullum et al., 2004) indicate RDX is unlikely to be found in urban environments, and air and consumer products are not likely to be a source for RDX exposure, human exposure is likely to be limited to water and soil contamination.

Physical-chemical properties of RDX (including its low water solubility, slow dissolution in aqueous solution, low vapor pressure, and low affinity for hydrophobic substances) predict it will have limited retention in soil or readily leach from soil (McGrath, 1995; Pennington et al., 1995; Cataldo et al., 1990), with the potential to migrate to groundwater. The potential to migrate to groundwater may suggest soil is not likely to be a significant source for RDX exposure, although soil could be a significant source in areas with little precipitation. Several studies identified indicate edible portions of garden and field crops have the potential to take up RDX from contaminated soil as well as from irrigation water. The concentration of RDX from soil and hydroponic media has been reported in plants (Harvey et al., 1991; Checkai and Simini, 1996;

Price et al., 1997). These results suggest food crops grown in RDX-contaminated soils or irrigated with RDX-contaminated water may be a source of RDX exposure.

This report demonstrates an approach that may be used to calculate RSCs in site-specific situations where national monitoring data are not available and national exposure is not anticipated, as is the case for RDX. The approach is based on the USEPA (2000) process that provides a framework to evaluate the adequacy of the available exposure data, exposure scenarios, and relevant sources of exposure. The USEPA process supports development of a chemical-specific RSC term for RDX of at least 50%.

A site-specific approach based on the assumption the relevant media will be remediated to a level dictated by a Superfund type risk assessment may also be used. In this approach, specific contaminated media are selected for remediation (e.g., soil and water) and the RSC can be assigned based on the level of clean-up determined. For example, RDX exposure may be apportioned at 50% RSC to each of the soil and water media. Safe concentrations (i.e., remediation goals) for each of the two media would then be calculated based upon appropriate exposure assumptions and values, and clean-up values are estimated based on these assignments. However, a different apportionment of the RDX exposures (i.e., RSC values) might be chosen if there are reasons (e.g., cost) to clean up one of the media more than another. For example, if RDX is more easily remediated from soil, then a higher RSC might be assigned to water. An example of this site-specific approach (i.e., using concentrations of RDX detected in soil, groundwater, surface water, sediment, etc.) to estimate intakes from these media as well as from food using equations discussed above is found in Appendix F.

## 9. References

- Agency for Toxic Substance and Disease Registry (ATSDR). 1995. *Toxicology Profile for RDX*. Atlanta, Ga.: Agency for Toxic Substances and Disease Registry, Public Health Service, US Department of Health and Human Services (HHS). Available at: <http://www.atsdr.cdc.gov/toxprofiles/tp78.html>.
- Belden, J.B., Lotufo, G.R., Lydy, M.J. 2005. Accumulation of Hexahydro-1,3,5-Trinitro-1,3,5-Triazine in Channel Catfish (*Ictalurus punctatus*) and Aquatic Oligochaetes (*Lumbriculus variegates*). *Environmental Toxicology and Chemistry*. 24(8):1962–1967.
- Bently, R.E., Dean, J.W., Ellie, S.J., Hollister, T.A., LeBlanc, G.A., Sauter, S., Sleight, B.H. 1977. *Laboratory evaluation of the toxicity of cyclotrimethylene trinitramine (RDX) to aquatic organisms*. AD A061730. Final Report. EG&G Bionomics, Wareham, Mass., for the US Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, Md.
- Bishop, R.W., Kennedy, J.L., Podolak, G.E., et al. 1988. A field evaluation of air sampling methods for TNT and RDX. *Journal of American Industrial Hygiene Association*. 49:635-638. [Cited in ATSDR, 1995].
- Burrow, E.P., Rosenblatt, D.H., Mitchell, W.R., Parmer, D.L. 1989. *Organic explosives and related compounds: environmental and health considerations*. AD-A210 554. US Army Biomedical Research and Development Laboratory, Fort Detrick, Md.
- Burton, D.T., Turley, D.S. 1995. Reduction of Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) Toxicity to the Cladoceran *Ceriodaphnia dubia* Following Photolysis in Sunlight. *Bulletin of Environmental Contamination and Toxicology*. 55:85–95.
- Cataldo, D.A., Harvey, S.D., Fellows, R.J. 1990. *An evaluation of the environmental fate and behavior of munitions materiel (TNT, RDX) in soil and plant systems*. *Environmental Fate and Behavior of RDX*. PNL-7529. Prepared for the US Army Biomedical Research and Development Laboratory, Fort Detrick, Fredrick, Md.
- Checkai, R.T., Simini, M. 1996. *Phytophysiological Response of Crops to Irrigation Waters Containing Low Concentrations of RDX and TNT: Ecotoxicological Implications*. Presented at 20<sup>th</sup> Army Science Conference, Science and Technology for Force XXI, 24–27 June 1996, Norfolk, Va.
- Checkai, R.T., Simini, M., Harvey, S.D. 1996. *Plant Uptake of RDX and TNT Utilizing Site Specific Criteria for the Cornhusker Army Ammunition Plant, Neb.* US Army ERDEC Technical Report. Project Order No. 560786M8AA.
- Cholakakis, J.M., Wong, L.C.K., Van Goethem, D.L., Minor, J., Short, R., Sprinz, H., Ellis, H.V. III. 1980. *Mammalian toxicological evaluation of RDX*. Report AD A092531. Prepared by the Midwest Research Institute, Kansas City, Mo., for the USAMRDC, Frederick, Md.
- Clausen, J.L., Scott, C., Cramer, R.J. 2007. *Development for environmental data for Navy, Air Force, and Marine munitions*. *Strategic Environmental Research and Development Program*. Report No. ER-1480. Cold Regions Research and Engineering Laboratory.
- Crouse, L.C.B., Michie, M. W., Major M. A., Leach G. and Reddy, G. 2008. Oral bioavailability



- of cyclotrimethylenetrinitramine (RDX) from contaminated site soils in rats. *International Journal of Toxicology*. 27:317–322.
- Crowson, A., Cullum, H.E., Lowe, A.M. 1996. A survey of high explosive traces in public places. *Journal of Forensic Science*. 41(56):980989.
- Cullum, H.E., McGavigan, C., Uttley, C.Z., Stroud, M.A.M., Warren, D.C. 2004. A Secondary survey of high explosive traces in public places. *Journal of Forensic Science*. 49(4):684–690.
- Dontsova, K.M., Yost, S.L., Simunek, J., Pennington, J.C., Williford, C.W. 2006. Dissolution and Transport of TNT, RDX, and Composition B in Saturated Soil Columns. *Journal of Environmental Quality*. 35:2043–2054.
- ENSR Consulting and Engineering. 1991. *Analytical results of soil, biota, and groundwater sampling at residences in capitol Heights and Leheights Subdivisions, Grand Island, Neb..* Document no. 6583-033-400. US Army Toxic and Hazardous Materials Agency. Aberdeen Proving Ground, Md. [Cited in Checkai et al., 1996].
- Etnier, E.L., and W.R. Hartley. 1990. Comparison of water quality criterion and lifetime health advisory for hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX). *Regulatory Toxicology and Pharmacology*. 11(2):118–122.
- Envirodyne Engineers, Inc. 1980. *Milan Army Ammunition Plant contamination survey. Final report*. AD-B053362. Envirodyne Engineers, Inc., St. Louis, Mo. [Cited in Talmage et al., 1999].
- Fellows, R.J., Harvey, S.D., Cataldo, D.A. 1995. *Evaluation of the metabolic fate of munitions material (TNT, RDX) in plant systems and initial assessment of material interaction with plant genetic material: Validation of the metabolic fate of munitions material (TNT, RDX) in mature crops*. PNL-10825/UC-402. Pacific Northwest Laboratories, Richland, Wash. [Cited in McKone and Maddalena, 2007].
- Fellows, R.J., Driver, C.R., Cataldo, D.A., Harvey, S.D. 2006. Bioavailability of hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) to the prairie vole (*Microtus ochrogaster*). *Environmental Toxicology and Chemistry*. 25(7):1881–18.
- Harvey, S.D., Fellows, R.J., Cataldo, D.A., Bean, R.M. 1991. Fate of the Explosive Hexahydro-1,3,5- Trinitro-1,3,5-Triazine (RDX) in Soil and Bioaccumulation in Bush Bean Hydroponic Plants. *Environmental Toxicology and Chemistry*. 10:845–855.
- Hoffsommer, J.C., Rosen, J.M. 1973. Hydrolysis of Explosives in Sea Water. *Bulletin of Environmental Contamination and Toxicology*. 10:78–79.
- Hollander, A.I., Colbach, E.M. 1969. Composition C-4 induced seizures: A report of five cases. *Military Medicine*. 134:1529–1530.
- Hazardous Substances Data Bank (HSDB). 1994. NLM, National Toxicology Information Program, Bethesda, Md. Available at: <http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB>. May 1994. [As cited by ATSDR 2005] [No longer online].
- HSDB. 2009. NLM, National Toxicology Information Program, Bethesda, Md. Available at: <http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB>. Accessed January 22, 2009.

- Jenkins, T.F., Hewitt, A.D., Ranney, T.A., Ramsey, C.A., Lambert, D.J., Bjella, K.L., Perron, N.M. 2005. "Sampling strategies near a low-order detonation and a target at an artillery impact area," Chapter 2, Distribution and fate of energetics on DoD test and training ranges: Report 5, ERDC TR-05-2, US Army Engineer Research and Development Center, Vicksburg, Miss.
- Jenkins, T.F., Hewitt, A.D., Grant, C.L., Thiboutot, S., Ampleman, G., Walsh, M.E., et al. 2006. Identity and distribution of residues of energetic compounds at Army live-fire training ranges. *Chemosphere*. 63:1280–1290.
- Kaplan, A.S., Berghout, C.F., Peczenik, A. 1965. Human intoxication from RDX. *Archives of Environmental Health*. 10:877–883.
- Ketel, W.B., Hughes, J.R. 1972. Toxic encephalopathy with seizures secondary to ingestion of an explosive material composition C-4: A clinical and electroencephalographic study. *Neurology*. 22:871–876.
- Krishnan, K., Crouse, L.C.B., Bazar, M.A., Major, M.A., Reddy, G. In Press. Physiologically based pharmacokinetic modeling of cyclotrimethylenetrinitramine in male rats. *Journal of Applied Toxicology*. 2009.
- Lachance, B., Rocheleau, S., Hawari, J., Gong, P., Leduc, F., Apte, J., Sarrazin, M., Martel, M., Bardai, G., Dodard, S., Sunahara, G.I. 2003. *Bioaccumulation of nitro-heterocyclic and nitroaromatic energetic materials in terrestrial receptors in a natural sandy loam soil*. Biotechnology Research Institute. National Research Council Canada.
- Larson, S.L., Jones, R.P., Escalon, L., Parker, D. 1999. Classification of explosives transformation products in plant tissue. *Environmental Toxicology and Chemistry*. 18(6):1270–1276.
- Layton, D., Mallon, B., Mitchell, W., Hall, L., Fish, R., Perry, L., Snyder, G., Bogen, K., Malloch, W., Ham, C., Dowd, P. 1987. *Conventional weapons demilitarization: a health and environmental effects data base assessment. Explosives and their co-contaminants. Final report, phase II*. AD-A220588. Lawrence Livermore National Laboratory, Livermore, Calif. USAMRDC, Frederick, Md.
- Liu, D.H., Bailey, H.C., Pearson, J.G. 1983. *Toxicity of a complex munitions wastewater to aquatic organisms*. In: Bishop, W.E., Cardwell, R.D., Heidolph, B.B. (eds) *Aquatic Toxicology and Hazard Assessment: Sixth Symposium*. ASTM STP 802. American Society for Testing and Materials, Philadelphia, Pa., 135–150.
- McCormick, N.G., Cornell, J.H., Kaplan, A.M. 1981. Biodegradation of hexahydro-1,3,5-trinitro-1,3,5-triazine. *Applied Environmental Microbiology*. 42(5):817–823.
- McGrath C.J. 1995. *Review of formulations for processes affecting the subsurface transport of explosives*. Technical Report IRRP-95-2. US Army Engineer Waterways Experiment Station, Vicksburg, Miss. Available online at: <http://el.erd.c.usace.army.mil/elpubs/pdf/trirrp95-2/trirrp952.pdf>.
- McKone, T.E., Maddalena, R.L. 2007. Plant uptake of organic pollutants from soil: Bioconcentration estimates based on models and experiments. *Environmental Toxicology and Chemistry*. 26(12):2494–2504.

- Merck. 1989. *The Merck index: An encyclopedia of chemical, drugs, and biologicals*. 11th Ed. Budavari, S., O'Neil, M.J., Smith, A., et al., eds. Rahway, N.J.: Merck & Co., Inc.
- Merrill, S.L. 1968. Ingestion of an explosive material, composition C-4: A report of two cases. *USARV Medical Bulletin* 3:5.
- National Fireworks Association (NFA). 2008. Schneider, R.L. 2009. Personal communication.
- National Institute of Health (NIH). 2008. Household Products Database. NLM Specialized Information Services, HHS. Available at: <http://hpd.nlm.nih.gov/>.
- National Occupational Exposure Survey (NOES). 1990. NOES (1981–83): RDX. HHS, NIOSH, Cincinnati, Ohio. NRC. 1982. Evaluation of the health risks of ordnance disposal waste in drinking water.
- Parker, G.A., Reddy, G., Major, M.A. 2006. Reevaluation of a twenty-four-month chronic toxicity/carcinogenicity study of hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) in the B6C3F1 hybrid mouse. *International Journal of Toxicology*. 25:373–378.
- Pennington, J.C., Myers, T.E., Davis, W.M., Olin, T.J., McDonald, T.A., Hayes, C.A., Townsend, D.M. 1995. *Impacts of Sorption on in Situ Bioremediation of Explosives-Contaminated Soils*. Technical Report IRRP-95-1. USACE, Waterways Experiment Station, Vicksburg, Miss.
- Pennington, J.C., Jenkins, T.F., Thiboutot, S., Ampleman, G., Clausen, J., Hewitt, A.D., Lewis, J., Walsh, M.R., Walsh, M.E., Ranney, T.A., Silverblatt, B., Marois, A., Gagnon, A., Brousseau, P., Zufelt, J.E., Poe, K., Bouchard, M., Martel, R., Walker, D.D., Ramsey, C.A., Hayes, C.A., Yost, S.L., Bjella, K.L., Trepanier, L., Berry, T.E., Lambert, D.J., Dubé, P., Perron, N.M. 2005. *Distribution and fate of energetics on DoD test and training ranges: Interim report 5*. Strategic Environmental Research and Development Program. Report No. ERDC TR-05-2.
- Price, R.A., Pennington, J.C., Neumann, D., Hayes, C.A., Larson, S.L. 1997. *Plant Uptake of Explosives from Contaminated Soil and Irrigation Water at the Former Nebraska Ordnance Plant, Mead, Nebraska*. Technical Report EL-97-11. US Army Engineer Waterways Experiment Station, Vicksburg, Miss.
- Reddy, G., Allen, N.A., Major, M.A. 2008. Absorption of <sup>14</sup>C-cyclotrimethylenetrinitramine (RDX) from soils through excised human skin. *Toxicology Mechanisms and Methods*. 18: 575–579.
- Reifenrath, W.G., Kammen, H.O., Palmer, W.G., Major, M.A., Leach, G.J. 2002. Percutaneous absorption of explosives and related compounds: An empirical model of bioavailability of organic nitro compounds from soil. *Toxicology and Applied Pharmacology*. 182:160–168.
- Reifenrath, W.G., Kammen, H.O., Reddy, G., Major, M.A., Leach, G.J. 2008. Interaction of hydration, aging, and carbon content of soil on the evaporation and skin bioavailability of munition contaminants. *Journal of Toxicology Environmental Health*. Part A, 71:486–494.

- Ringelberg, D.B., Reynolds, C.M., Walsh, M.E., Jenkins, T.F. 2003. RDX Loss in a Surface Soil under Saturated and Well Drained Conditions. *Journal of Environmental Quality*. 32:1244–1249.
- Schneider, N.R., Bradley, S.L., Anderson, M.E. 1977. Toxicology of cyclotrimethylenetrinitramine: Distribution and metabolism in the rat and the miniature swine. *Toxicology and Applied Pharmacology*. 39:531–541.
- Schneider, N.R., Bradley, S.L., Anderson, M.E. 1978. The distribution and metabolism of cyclotrimethylenetrinitramine (RDX) in the rat after subchronic administration. *Toxicology and Applied Pharmacology*. 46:163–171.
- Small, M.J., and Rosenblatt, D.H. 1974. *Munitions production products of potential concern as waterborne pollutants: Phase II*. AD-919031. US Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, Md. [Cited in Talmage et al., 1999].
- Spalding, R.F., Fulton, J.W. 1988. Groundwater munition residues and nitrate near Grand Island, Neb. USA. *Journal of Contaminant Hydrology*. 2:139–153. [Cited in Talmage et al., 1999].
- Spanggord, R.J., Mabey, W.R., Mill, T., et al. 1981. *Environmental fate studies on certain munition wastewater constituents: Phase 3, part 2*. Laboratory studies. NTIS AD-A131 90. Menlo Park, Calif.: SRI International, 58. [Cited in Talmage et al., 1999].
- Stone, W.J., Paletta, T.L., Heiman, E.M., et al. 1969. Toxic effects following ingestion of C-4 plastic explosive. *Archives of Internal Medicine*. 124:726–730.
- Talmage, S.S., Opresko, D.M., Maxwell, C.J., et al. 1999. Nitroaromatic Munition Compounds: Environmental Effects and Screening Values. *Reviews of Environmental Contamination and Toxicology*. 161:1–156.
- Turley, C.P., Brewster, M.A. 1987. Liquid-chromatographic analysis of cyclotrimethylenetrinitramine in biological fluids using solid-phase extraction. *Journal Chromatography and Biomedical Applications*. 421:430–433.
- Twibell, J.D., Turner, S.L., Smalldon, K.W., et al. 1984. The persistence of military explosives on hands. *Journal of Forensic Science*. 29:284–290. [Cited in ATSDR, 1995].
- US Army. 1975. *Development of industrial hygiene sampling and analytical methodology for evaluation of exposures to TNT and associated explosives*. Contract no. DADA17-73-C-3167. Washington, D.C.: USAMRDC. Document No. AD-A008 399. (author: Saltzman, B.E. et al.) [Cited as Army 1975 in ATSDR, 1995].
- US Army. 1984. *Database assessment of the health and environmental effects of munition production waste products*. Frederick, Md.: USAMRDC, Fort Detrick. Document no. AD-A145 417. [Authored by Ryon M.G. et al.). [Cited as Army 1984a in ATSDR, 1995].
- US Army Corps of Engineers (USACE). 2002. *Identity and Distribution of Residues of Energetic Compounds at Military Live-Fire Training Ranges*. Engineering, Research, and Development Center. ERDC TR-05-10.
- USACE. 2005. *Identity and Distribution of Residues of Energetic Compounds at Military Live-Fire Training Ranges*. Engineering, Research, and Development Center. ERDC TR-05-10.

- US Army Center for Health Promotion and Preventive Medicine (USACHPPM). 1994. *Health risk assessment for consumption of deer muscle and liver from Joliet Army Ammunition Plant, Joliet, Illinois. Final Report*. Project No. 75-51-YF23. Formerly US Army Environmental Hygiene Agency. Aberdeen Proving Ground, Md.
- USACHPPM. 2002. *Bioconcentration, bioaccumulation, and biomagnification of nitroaromatic and nitramine explosives and their breakdown products*. Toxicology Report No. 87-MA-4677-01. USACHPPM, Aberdeen Proving Ground, Md.
- USACHPPM. 2006. *Bioconcentration, Bioaccumulation, and Biomagnification of Nitroaromatic and Nitramine Explosives and their Breakdown Products. Toxicology Study No. 87-MA-4677-01*. USACHPPM, DoD. 1976. AD-A040-161. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexandria, VA, 22314, or call (703) 274-7633.
- US Army Medical Research and Development Command (USAMRDC). 1980. *Environmental Fate of RDX and TNT*. DoD, Technical Report. 81-538.
- USAMRDC. 1983. *Environmental Fate Studies of HMX: Phase II – Detailed Studies*. Report by SRI International, Menlo Park, Calif.
- US Department of Defense (US DoD). 1983. *Determination of the Chronic Mammalian Toxicological Effects of RDX: Twenty-four Month Chronic Toxicity/Carcinogenicity Study of Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) in the Fischer 344 Rat. Phase V., Vol. 1*. AD A160774. IIT Research Institute, Chicago, Ill. USAMRDC. Report No. AD-A160-774.
- US DoD. 1984. *Determination of the Chronic Mammalian Toxicological Effects of RDX: Twenty-four Month Chronic Toxicity/Carcinogenicity Study of Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) in the B6C3F1 Hybrid Mouse. Phase VI., Vol. 1*. AD A160774. IIT Research Institute, Chicago, Ill. Available from USAMRDC, Ft. Detrick, Frederick, Md. 21701.
- US Environmental Protection Agency (USEPA). 1986. *Guidelines for Carcinogen Risk Assessment*. National Center for Environmental Assessment. Published on September 24, 1986, Federal Register 51(185):33992-34003.
- USEPA. 1988. *Health Advisory for Hexahydro-1,3,5-Trinitro-1,3,5-Triazine (RDX)*. Office of Drinking Water, Washington, D.C., November. National Technical Information Service (NTIS) PB90-273533.
- USEPA. 1989. *Risk Assessment Guidance for Superfund, Volume 1, Part A*. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-89/002.  
<http://www.epa.gov/oswer/riskassessment/ragsa/index.htm>.
- USEPA. 1991. *Risk Assessment Guidance for Superfund, Volume 1, Part B*. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/R-92/003.  
<http://www.epa.gov/oswer/riskassessment/ragsb/>.
- USEPA. 2000. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health*. USEPA Office of Water, Office of Science and Technology, EPA-822-B-00-005.

- USEPA. 2005. UCMR for Public Water Systems Revisions. 70 Federal Register 49093 (Aug. 22, 2005); available from <http://www.epa.gov/safewater/ucmr/index.html>.
- USEPA. 2008. IRIS. National Center for Environmental Assessment (NCEA). Available at: <http://cfpub.epa.gov/ncea/iris/index.cfm>.
- USEPA. 2009a. Pesticide Product Information System (PPIS). Office of Pesticides Program (OPP). Available at: <http://www.epa.gov/opppmsd1/PPISdata/>.
- USEPA. 2009b. IRIS Track Report for Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) Assessment. IRIS Substance Assessment Tracking System (IRIS Track). Available at: [http://cfpub.epa.gov/ncea/iristrac/index.cfm?fuseaction=viewChemical.showChemical&sw\\_id=1089&msc=ai](http://cfpub.epa.gov/ncea/iristrac/index.cfm?fuseaction=viewChemical.showChemical&sw_id=1089&msc=ai).
- US Food and Drug Administration (US FDA). 2004. *Total Diet Study Market Baskets 1991–3 through 2003–4*. Center for Food Safety and Applied Nutrition, College Park, Md. Available at: <http://www.cfsan.fda.gov/~acrobat/tds1byps.pdf>.

**Appendix A**  
**Regulations and Guidelines Applicable to RDX (as of December 2008)**

AGENCY/ ORGANIZATION	DESCRIPTION	STANDARD OR GUIDELINE	REFERENCE
<b>Federal Standards and Guidelines</b>			
American Conference of Industrial Hygienists	Threshold Limit Value (TLV)	0.5 mg/m <sup>3</sup> (8-hour TWA) based on liver damage (1994)	ACGIH 2008 TLVs <sup>®</sup> and BEIs <sup>®</sup>
	Skin Notation	Potential for significant contribution to the overall exposure by the cutaneous route, including mucous membranes and the eyes, by contact with vapors, liquids, and solids.	
	Carcinogenicity	A4—Not Classifiable as a Human Carcinogen (1994)	
Agency for Toxic Substances and Disease Registry	Minimal Risk Levels (MRLs) -- an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure.	Oral MRLs: – Acute exposure duration (1-14 days) <ul style="list-style-type: none"> <li>▪ MRL = 0.06 mg/kg/day (uncertainty factor = 100)</li> <li>▪ Endpoint = neurological</li> </ul> – Intermediate exposure duration (>14-364 days) <ul style="list-style-type: none"> <li>▪ MRL = 0.03 mg/kg/day (uncertainty factor = 300)</li> <li>▪ Endpoint = reproductive</li> </ul> (September 1997 data)	<a href="http://www.atsdr.cdc.gov/mrls/index.html#bookmark01">http://www.atsdr.cdc.gov/mrls/index.html#bookmark01</a>
	CERCLA Priority List of Hazardous Substances	RDX ranks as #92 (out of 275) on the 2007 CERCLA Priority List of Hazardous Substances	<a href="http://www.atsdr.cdc.gov/cercla/07list.html">http://www.atsdr.cdc.gov/cercla/07list.html</a>
Department of Homeland Security	Chemicals of Interest	RDX and RDX/HMX mixtures are listed as DHS Chemicals of Interest.	6 CFR 27, Appendix A



AGENCY/ ORGANIZATION	DESCRIPTION	STANDARD OR GUIDELINE	REFERENCE
Department of Transportation	Hazardous Materials	<ul style="list-style-type: none"> <li>– Designated as a hazardous substance subject to requirements for Packaging, labeling, and transportation.</li> <li>– Class 1.1D explosive - domestic transportation limited to roads and water.</li> </ul>	49 CFR 172.101, Appendix A; 49 CFR 173.
Environmental Protection Agency	Contaminant Candidate List (CCL)	Included on the CCL 1 – but not selected for regulatory determination.	<p>Announcement of the Drinking Water Contaminant Candidate List (Notice), 63 Fed. Reg. 10273 (March 2, 1998).</p> <p>Announcement of Regulatory Determinations for Priority Contaminants on the Drinking Water Contaminant Candidate List (Notice). 68 Fed. Reg. 42897 (July 18, 2003).</p>
		Included on the CCL 2 (carried forward from CCL 1) – but not selected for regulatory determination.	<p>Drinking Water Contaminant Candidate List 2 (Final Notice), 70 Fed. Reg. 9071 (February 24, 2005).</p> <p>Drinking Water: Regulatory Determinations Regarding Contaminants on the Second Drinking Water Contaminant Candidate List—Preliminary Determinations (Notice), 72 Fed. Reg. 24015 (May 1, 2007).</p>
		<p>Included on the Draft CCL3. Public comment period closed May 21, 2008.</p> <p>Most recent activity on the Draft CCL3 is that the USEPA SAB met on October 28, 2008 to review their draft report, <i>Advisory on Contaminant Candidate List 3</i>. See Science Advisory Board Staff Office Notification of a Meeting of the Science Advisory Board, 73 Fed. Reg. 55512, September 25, 2008.</p>	Drinking Water Contaminant Candidate List 3—Draft (Notice), 73 Fed. Reg. 9627 (February 21, 2008).



AGENCY/ ORGANIZATION	DESCRIPTION	STANDARD OR GUIDELINE	REFERENCE
	Unregulated Contaminant Monitoring Regulation (UCMR)	<p>Included in the UCMR2 list of contaminants for which assessment monitoring is required. Monitoring will occur during 2008-2010. Approved analytical method is EPA Method 529 (SPE/GC/MS).</p> <p>Note that RDX was previously included on the UCMR1 (1999) list but not monitored because the analytical method required further refinement at the time the rule was implemented. (UCMR (1999) List 1 and List 2 Chemical Analytical Methods and Quality Control Manual. USEPA Office of Water, EPA-815-R-01-028. December 2001).</p>	Unregulated Contaminant Monitoring Regulation (UCMR) for Public Water Systems Revisions (Final Rule), 72 Fed. Reg. 368 (January 4, 2007).
	Drinking Water Health Advisories	<ul style="list-style-type: none"> <li>– One-day = 0.1 mg/L (10-kg child)</li> <li>– Ten-day = 0.1 mg/L (10-kg child)</li> <li>– Lifetime = 0.002 mg/L (70-kg adult)</li> <li>– RfD = 0.003 mg/kg/day</li> <li>– DWEL = 0.1 mg/L</li> <li>– mg/L at 10<sup>-4</sup> cancer risk = 0.03</li> <li>– Cancer descriptor = C (possible human carcinogen)</li> </ul>	<p>2006 Edition of the Drinking Water Standards and Health Advisories. EPA 822-R-06-013. USEPA Office of Water (Summer 2006).</p> <p><a href="http://www.epa.gov/waterscience/criteria/drinking/dwstandards.pdf">http://www.epa.gov/waterscience/criteria/drinking/dwstandards.pdf</a>.</p>
	High Production Volume (HPV) Challenge Program	<p>RDX is classified as an HPV chemical, but was not sponsored by either a company or international effort for the development of health and environmental effects data. As a result, it is considered an "orphan" chemical.</p> <p>The USEPA relied on TSCA Section 8(a) and 8(d) regulatory efforts to gather information on RDX.</p>	<p>Preliminary Assessment Information Reporting (PAIR); Addition of Certain Chemicals (Final Rule and Technical Corrections), 71 Fed. Reg. 47122 (August 16, 2006).</p> <p>Health and Safety Data Reporting; Addition of Certain Chemicals (Final Rule and Technical Corrections), 71 Fed. Reg. 47130 (August 16, 2006).</p>

AGENCY/ ORGANIZATION	DESCRIPTION	STANDARD OR GUIDELINE	REFERENCE
	Integrated Risk Information System (IRIS) - Health assessment toxicity benchmarks	<ul style="list-style-type: none"> <li>– Oral RfD = <math>3 \times 10^{-3}</math> mg/kg/day (UF = 100; MF = 1) based on prostate inflammation observed in a two-year rat feeding study from which an NOEL of 0.3 mg/kg/day and an LOAEL of 1.5 mg/kg/day was calculated<sup>1</sup>.</li> <li>– Inhalation RfC -- not available.</li> <li>– Carcinogenicity classification = C (possible human carcinogen) based on hepatocellular adenomas and carcinomas in female B6C3F1 mice.<sup>2</sup></li> <li>– Carcinogenic risk from oral exposure: <ul style="list-style-type: none"> <li>▪ Oral slope factor = <math>1.1 \times 10^{-1}</math> per (mg/kg)/day</li> <li>▪ Drinking water unit risk = <math>3.1 \times 10^{-6}</math> per (µg/L)</li> </ul> </li> <li>– Carcinogenic risk from inhalation exposure - not available.</li> </ul> <p>(IRIS data last revised February 1, 1993 (oral reference dose assessment) and July 1, 1993 (carcinogenicity assessment)).</p>	<a href="http://www.epa.gov/ncea/iris/subst/0313.htm">http://www.epa.gov/ncea/iris/subst/0313.htm</a>
	Manufacturer Effluent Guidelines and Standards	Explosive manufacturers (e.g., RDX, HMX, TNT, dynamite, nitroglycerin) shall meet point source effluent limitations for chemical oxygen demand, biological oxygen demand (5-day), total suspended solids and pH.	40 CFR 457

<sup>1</sup> U.S. DoD, 1983 - get reference from IRIS.

<sup>2</sup> U.S. DoD, 1984 - get reference from IRIS.

AGENCY/ ORGANIZATION	DESCRIPTION	STANDARD OR GUIDELINE	REFERENCE
EPA Regional Offices	Risk Based Concentrations (RBCs) (EPA Region 3)	<p>Screening Levels (SLs):</p> <ul style="list-style-type: none"> <li>– Residential Soil = 5.5 mg/kg (where noncancer SL &lt; 100X cancer SL)</li> <li>– Industrial Soil = 24 mg/kg (cancer)</li> <li>– Tapwater = 0.61 µg/L (cancer)</li> <li>– Risk Based Soil SL for Protection of Groundwater = <math>3.6 \times 10^{-4}</math> mg/kg</li> </ul> <p>Based on:</p> <ul style="list-style-type: none"> <li>– Oral Slope Factor (SFO) = <math>0.11 \text{ (mg/kg-day)}^{-1}</math> (IRIS)</li> <li>– Oral Reference Dose (RfDo) = <math>3 \times 10^{-3}</math> mg/kg-day (IRIS)</li> <li>– Risk Assessment Guidance (RAGS) Part E dermal absorption from soil (ABS) = 0.015</li> </ul>	<a href="http://www.epa.gov/reg3hwmd/risk/human/index.htm">http://www.epa.gov/reg3hwmd/risk/human/index.htm</a> (September 2008)
	Human Health Media-Specific Screening Levels (HHMSSLs) (Region 6)	Region 6 HHMSSLs have been harmonized with the Region 3 RBCs and the Region 9 RSLs. See values above.	<a href="http://www.epa.gov/region6/6pd/rcra_c/pd-n/screen.htm">http://www.epa.gov/region6/6pd/rcra_c/pd-n/screen.htm</a> (January 2009)
	Regional Screening Levels (RSLs) (EPA Region 9 – previously Preliminary Remediation Goals or PRGs)	Region 9 PRGs have been harmonized with the Region 3 RBCs -- see values above.	<a href="http://www.epa.gov/region09/superfund/prg/index.html">http://www.epa.gov/region09/superfund/prg/index.html</a> (September 2008)
National Institute for Occupational Safety and Health	Recommended Exposure Limit (REL)	1.5 mg/m <sup>3</sup> (10-hour TWA)	<p>NIOSH Pocket Guide to Chemical Hazards, CDC NIOSH, September 2005.</p> <p><a href="http://www.cdc.gov/niosh/npg/default.html">http://www.cdc.gov/niosh/npg/default.html</a></p>
	Short Term Exposure Limit (STEL)	3 mg/m <sup>3</sup> (15-minute TWA)	
	Skin Designation	Indicates the potential for dermal absorption; skin exposure should be prevented as necessary through the use of good work practices, gloves, coveralls, goggles, and other appropriate equipment.	
Occupational Safety and Health Administration	Permissible Exposure Limit and Skin Designation	Vacated	Air Contaminants (Final Rule), 58 Fed. Reg. 35338 (June 30, 1993).

AGENCY/ ORGANIZATION	DESCRIPTION	STANDARD OR GUIDELINE	REFERENCE
<b>State Standards and Guidelines</b>			
Alabama	Preliminary Screening Values	Direct Contact Exposure Pathways: <ul style="list-style-type: none"> <li>– Groundwater/Tap Water: <math>6.10 \times 10^{-4}</math> mg/L</li> <li>– Residential Soil: 4.40 mg/kg</li> <li>– Commercial Soil: <math>1.60 \times 10^{+1}</math> mg/kg</li> </ul>	Alabama Risk-Based Corrective Action Guidance Manual, April 2008, Revision 2, Alabama Department of Environmental Management. <a href="http://www.adem.state.al.us/LandDivision/Guidance/ARBCAApril2008final.pdf">http://www.adem.state.al.us/LandDivision/Guidance/ARBCAApril2008final.pdf</a>
Florida	Cleanup Target Levels (CTLs)	Following CTLs are based on reproductive effects and carcinogenicity. <ul style="list-style-type: none"> <li>– Groundwater and Surface Water:               <ul style="list-style-type: none"> <li>▪ Groundwater criteria = 0.3 µg/L</li> <li>▪ Freshwater surface water criteria = 180 µg/L</li> <li>▪ Marine surface water criteria = 180 µg/L</li> <li>▪ Groundwater of low yield / poor quality = 3 µg/L</li> </ul> </li> <li>– Soil:               <ul style="list-style-type: none"> <li>▪ Direct exposure - residential = 7.7 mg/kg</li> <li>▪ Direct exposure - commercial/industrial = 28 mg/kg</li> <li>▪ Leachability based on groundwater criteria = 0.002 mg/kg</li> <li>▪ Leachability based on freshwater surface water criteria = 1.3 mg/kg</li> <li>▪ Leachability based on marine surface water criteria = 1.3 mg/kg</li> <li>▪ Leachability based on groundwater of low yield / poor quality = 0.02 mg/kg</li> </ul> </li> </ul>	F.A.C. 62-777.170
	Natural Attenuation Default Concentrations	Groundwater criteria = 0.3 µg/L Natural attenuation default source = 30 µg/L	F.A.C. 62-777.170

AGENCY/ ORGANIZATION	DESCRIPTION	STANDARD OR GUIDELINE	REFERENCE
Illinois	Aquatic Life Criteria	<ul style="list-style-type: none"> <li>– Acute = 2 mg/L</li> <li>– Chronic = 0.5 mg/L</li> </ul>	<p>Date derived - January 5, 1993.</p> <p><a href="http://www.epa.state.il.us/water/water-quality-standards/water-quality-criteria-list.pdf">http://www.epa.state.il.us/water/water-quality-standards/water-quality-criteria-list.pdf</a></p>
Iowa	Standards for Groundwater, Iowa Land Recycling Program	<ul style="list-style-type: none"> <li>– Statewide Standard for Protected Groundwater: 0.002 mg/L</li> <li>– Statewide Standard for Non-Protected Groundwater Source: 0.032 mg/L</li> <li>– Statewide Standard for Soil: 22 mg/kg</li> </ul>	<p>Statewide Standards for Contaminants in Soil and Groundwater (unknown date)</p> <p><a href="http://programs.iowadnr.gov/riskcalc/pages/standards.aspx">http://programs.iowadnr.gov/riskcalc/pages/standards.aspx</a></p>
Kansas	Tier 2 Risk-Based Standards	<ul style="list-style-type: none"> <li>– Residential Scenarios <ul style="list-style-type: none"> <li>▪ Soil pathway = 44 mg/kg</li> <li>▪ Soil to groundwater protection pathway = 0.13 mg/kg</li> <li>▪ Groundwater pathway = 0.008 mg/L</li> </ul> </li> <li>– Non-residential Scenarios <ul style="list-style-type: none"> <li>▪ Soil pathway = 44 mg/kg</li> <li>▪ Soil to groundwater protection pathway = 0.43 mg/kg</li> <li>▪ Groundwater pathway = 0.03 mg/L</li> </ul> </li> </ul> <p><i>Note: groundwater pathway based on carcinogenic risk of <math>1 \times 10^{-5}</math>.</i></p>	<p>Risk-Based Standards for Kansas RSK Manual -- 4th Version, June 2007. Kansas Department of Health and Environment, Division of Environment, Bureau of Environmental Remediation.</p> <p><a href="http://www.kdheks.gov/remedial/rsk_manual_page.htm">http://www.kdheks.gov/remedial/rsk_manual_page.htm</a></p>

AGENCY/ ORGANIZATION	DESCRIPTION	STANDARD OR GUIDELINE				REFERENCE
Massachusetts	Massachusetts Contingency Plan (MCP) Method 1 Groundwater Standards	GW-1 = 1 µg/L				310 CMR 40.0974(2), Table 1 and 40.0932 (December 14, 2007)
		GW-2 = 50,000 µg/L				
		GW-3 = 50,000 µg/L				
		GW-1: Groundwater is located within a current or potential drinking water source area.				
		GW-2: Groundwater located within 30 ft of existing or planned building or structure that is or will be occupied, and the average annual depth to groundwater in that area is 15 ft or less. Considered to be a potential source of vapors of oil and/or hazardous material to indoor air.				
	GW-3: Groundwater at all disposal sites shall be considered a potential source of discharge to surface water and shall be categorized, at a minimum, as category GW-3.					
	MCP Method 1 Soil Category Standards Applicable to Soil for a Combination of Soil and Groundwater Categories	Groundwater Category	Soil Standard (ppm)			310 CMR 40.0975(6)(a), Tables 2, 3, and 4 (December 14, 2007)  310 CMR 40.0933 (December 14, 2007)
S-1 Soil			S-2 Soil	S-3 Soil		
GW-1		1	1	1		
GW-2		8	60	100		
GW-3		8	60	200		
Soil shall be classified as either category S-1, S-2 or S-3 – classification shall consider the site, receptor, and exposure information, considering current and reasonably foreseeable site activities and uses. The three soil categories describe a range of the potential for exposure to that soil: category S-1 soils are associated with the highest potential for exposure—category S-3 soils have the lowest potential for exposure.						
MCP Method 2 Direct Contact Exposure-Based Soil Concentrations	S-1 = 8 ppm S-2 = 60 ppm S-3 = 200 ppm				310 CMR 40.0985(6), Table 5 (December 14, 2007)	

AGENCY/ ORGANIZATION	DESCRIPTION	STANDARD OR GUIDELINE	REFERENCE
	Upper Concentration Limits	Groundwater = 100,000 µg/L Soil = 2,000 ppm	310 CMR 40.0996(7), Table 6 (December 14, 2007)
Mississippi	Tier 1 Target Remedial Goals	<ul style="list-style-type: none"> <li>Groundwater = 0.609 µg/L (carcinogenic endpoint)</li> <li>Soil (restricted) = 52 mg/kg (carcinogenic risk)</li> <li>Soil (unrestricted) = 5.81 mg/kg (carcinogenic risk)</li> </ul>	<p>Risk Evaluation Procedures for Voluntary Cleanup and Redevelopment of Brownfield Sites, Subpart II, Section 601.</p> <p><a href="http://www.deq.state.ms.us/newweb/MDEQRegulations.nsf?OpenDatabase">http://www.deq.state.ms.us/newweb/MDEQRegulations.nsf?OpenDatabase</a></p>
Missouri	Risk-Based Target Levels	<ul style="list-style-type: none"> <li>Lowest default target levels (all soil types and all pathways) <ul style="list-style-type: none"> <li>Soil = <math>6.5 \times 10^{-2}</math> mg/kg (protection of domestic groundwater use pathway)</li> <li>Groundwater = <math>6.07 \times 10^{-3}</math> mg/L (domestic water use of groundwater)</li> </ul> </li> <li>Additional values provided in Appendix B for specific pathways and soil types.</li> </ul>	<p>Missouri Risk-Based Corrective Action Technical Guidance, Appendix B (April 2006). Hazardous Waste Program, Missouri Department of Natural Resources</p> <p><a href="http://www.dnr.mo.gov/env/hwp/mrbca/mrbca.htm">http://www.dnr.mo.gov/env/hwp/mrbca/mrbca.htm</a></p>
Nebraska	Voluntary Cleanup Program Remediation Goals	<ul style="list-style-type: none"> <li>Direct Contact Exposure Pathways <ul style="list-style-type: none"> <li>Residential Soil 4.4 mg/kg</li> <li>Industrial Soil <math>1.6 \times 10^{+02}</math> mg/kg</li> <li>Groundwater <math>6.1 \times 10^{-01}</math> µg/L</li> </ul> </li> </ul>	<p>Nebraska Voluntary Cleanup Program Guidance, 05-162, Attachment A (October 2008). Nebraska Department of Environmental Quality Remediation Section</p> <p><a href="http://www.deq.state.ne.us/Publications/pages/05-162">http://www.deq.state.ne.us/Publications/pages/05-162</a></p>
New Jersey	Interim Groundwater Quality (GWQ) Criteria	<p>Interim GWQ Criterion = 0.3 ppb</p> <p>Practical Quantitation Limit (PQL) = 0.5 ppb</p> <p>The higher of the PQLs and interim criteria is the numerical standard to be applied for each constituent in Class II-A aquifers (N.J.A.C. 7:9C-1.9(c)).</p>	<p><a href="http://www.state.nj.us/dep/wms/bwgsa/gwqs_interim_criteria_table.htm">http://www.state.nj.us/dep/wms/bwgsa/gwqs_interim_criteria_table.htm</a></p> <p>(February 2008)</p>

AGENCY/ ORGANIZATION	DESCRIPTION	STANDARD OR GUIDELINE	REFERENCE
New Mexico	Soil Screening Levels	<ul style="list-style-type: none"> <li>Residential soil = 44.2 mg/kg (carcinogenic endpoint)</li> <li>Industrial / occupational soil = 174 mg/kg (carcinogenic endpoint)</li> <li>Construction worker soil = 699 mg/kg (non-carcinogenic endpoint)</li> <li>Tapwater = 6.03 µg/L (carcinogenic endpoint)</li> </ul>	<p>New Mexico Technical Background Document for Development of Soil Screening Levels, Revision 4.0, June 2006.</p> <p><a href="http://www.nmenv.state.nm.us/gwb/documents/NMED_June_2006_SSG.pdf">http://www.nmenv.state.nm.us/gwb/documents/NMED_June_2006_SSG.pdf</a></p>
Texas	Aquatic Life Surface Water Risk Based Exposure Limits ( <sup>SW</sup> RBEL)	<p>Freshwater acute <sup>SW</sup>RBEL = 1,080 µg/L</p> <p>Freshwater chronic <sup>SW</sup>RBEL = 180 µg/L</p>	<p>October 2005.</p> <p><a href="http://www.tceq.state.tx.us/assets/public/remediation/trrp/swrbelstable.doc">http://www.tceq.state.tx.us/assets/public/remediation/trrp/swrbelstable.doc</a></p>
	Tier 1 Sediment Protective Concentration Levels (PCLs)	<ul style="list-style-type: none"> <li>Combined (ingestion + dermal pathways) -- <math>\text{TotSed}_{\text{Comb}} = 130 \text{ mg/kg}</math> (based on carcinogenic effects)</li> <li>Carcinogenic <ul style="list-style-type: none"> <li><math>\text{TotSed}_{\text{Comb}} = 130 \text{ mg/kg}</math></li> <li><math>\text{Sed}_{\text{Ing}} = 500 \text{ mg/kg}</math></li> <li><math>\text{Sed}_{\text{Derm}} = 170 \text{ mg/kg}</math></li> </ul> </li> <li>Noncarcinogenic <ul style="list-style-type: none"> <li><math>\text{TotSed}_{\text{Comb}} = 460 \text{ mg/kg}</math></li> <li><math>\text{Sed}_{\text{Ing}} = 2,200 \text{ mg/kg}</math></li> <li><math>\text{Sed}_{\text{Derm}} = 580 \text{ mg/kg}</math></li> </ul> </li> </ul>	<p>Last update - March 31, 2006.</p> <p><a href="http://www.tceq.state.tx.us/assets/public/remediation/trrp/sedpcls_2006.pdf">http://www.tceq.state.tx.us/assets/public/remediation/trrp/sedpcls_2006.pdf</a></p>



**Appendix B**  
**Media Concentrations of RDX from 34 DOD Facilities\***

	Amount discharged from facility	Concentration in untreated wastewaters	Soil	Surface Water	Ground Water	Sediments	Comments
Holston AAP, Kingsport, TN	69.5 kg/day (in 1980) Spangford et al. (1980)	<0.005 (detection limit) – 4.75 mg/L (untreated wastewaters)  <0.05-0.7 mg/L (treated wastewaters)  Stillwell et al. (1977)	70-80 mg/kg Bender et al. (1977)				Holston AAP described as primary site of manufacture in 1977. The only U.S. facility engaged in the manufacture of RDX as of 1989 (Burrows et al., 1989).
Aberdeen Proving Ground	3000 L/year				0.3 mg/L (exposure point concentration in a ditch that fed into a local creek) USACHPPM (2005)		Oldest continuously operated military testing area in the US (USACHPPM 2005)
Bangor Naval Submarine Base, Hood Canal near Silverdale, WA							Soil remediated and ground water is being pumped and treated; control installed to prevent leaching. No concentrations reported in USACHPPM (2005)
Nansemond Ordnance Depot, VA			No RDX found (USACHPPM 2005)	No RDX found (USACHPPM 2005)	12.3 µg/L (max) (USACHPPM 2005)		Former US military facility.

	Amount discharged from facility	Concentration in untreated wastewaters	Soil	Surface Water	Ground Water	Sediments	Comments
Seneca Army Depot Activity (BRAC facility), NY			No RDX found USACHPPM (2005)	No RDX found USACHPPM (2005)	No RDX found USACHPPM (2005)		No concentrations reported for soil, ground or surface water
Sierra Army Depot, CA			5.79 – 8.34 mg/kg Walsh and Jenkins (1992)	No surface water exists on site	No RDX found (USA CHPPM 2005)		
Milan AAP, TN			139-616 mg/kg (mean, 378 mg/kg) (3/7 samples) Walsh and Jenkins (1992)  39 mg/kg Jenkins and Grant (1987)	0.1-109 mg/L (mean: 11.9 mg/L) (concentration entering stream on site) Spanggord et al. (1978)  <0.4 - 110 µg/L (stream water concentration) Envirodyne Engineers, Inc. (1980)  <4 - <1600 µg/L (pink water lagoon) Envirodyne Engineers, Inc. (1980)	<20 - 780 µg/L (below soil containing 0.05-83 mg/kg Envirodyne Engineers, Inc. (1980)*  30.0 mg/L (max concentration in GW at site) Tucker et al. (1985)	290-43,000 mg/kg Envirodyne Engineers, Inc. (1980)  2600 – 38,000 mg/kg (sediment concentration in pink water lagoon) Envirodyne Engineers, Inc. (1980)	

	Amount discharged from facility	Concentration in untreated wastewaters	Soil	Surface Water	Ground Water	Sediments	Comments
Iowa AAP		0.1 - 24 mg/L Spanggord et al. (1978)	97.4 - 13,900 mg/kg (median, 7000 mg/kg) (2/6 samples) Walsh and Jenkins (1992)	0.1 - 15 mg/L (Brush Creek) Small and Rosenblatt (1974)	36.0 mg/L (max. concentration) Tucker et al., (1985)  Up to 445 ppb USACHPPM (2005)  12,785±1,744 µg/L Best et al. (1997)		
Louisiana AAP			185 - 972 mg/kg (median, 578 mg/kg) (2/2 samples) Walsh and Jenkins (1992)	5.6 - 28.9 mg/L (inactive lagoon) Spanggord et al. (1983)	<0.1 – 14,120 µg/L (concentrations underlying several areas) Gregory and Elliott (1987)  13-27,000 µg/L (in 6 of 11 wells below an area of pink water leaching lagoons) Todd et al. (1989)  17.8 mg/L (max in GW) Tucker et al. (1985)	400 - 120,000 mg/kg (highest concentration within the top 0.05 m Spanggord et al. (1983)	

	Amount discharged from facility	Concentration in untreated wastewaters	Soil	Surface Water	Ground Water	Sediments	Comments
Cornhusker AAP (near Grand Island, Hall County, NB)					<p>Earlier than 1988 monitoring: 300 µg/L, onsite GW downgradient of site; &gt;35 µg/L, offsite plume concentration.</p> <p>1988 monitoring:</p> <p>Plume 6.5 km long and 1.6 km wide: offsite concentrations up to 100 µg/L; estimated transport velocity: 0.5 m/d. Spalding and Fulton (1988)</p>	2 - 40 mg/kg (sediments taken from leaching pits at the site) Rosenblatt (1986)	Highest RDX concentration detected in soil was 12,000 ppm. However, USACHPPM (2005) says soil excavated to 5 ft below the water table level but no post excavation soil samples could be taken and soil concentrations are assumed to be higher than the target levels
Savanna Army Depot						3000-4000 mg/kg (concentration at surface of a dry lagoon) Rosenblatt (1986)	

	Amount discharged from facility	Concentration in untreated wastewaters	Soil	Surface Water	Ground Water	Sediments	Comments
Lone Star AAP					<p>&lt;20 - &gt;700 µg/L (in the vicinity of a contaminated site) Burrows et al. (1989)</p> <p>1 – 47 µg/L (in 7 of 7 wells at an unidentified munitions disposal site) Richards and Junk (1986)</p>	<p>Up to 50,000 mg/kg (sludge below pink water settling points) Phung and Bulot (1981)</p> <p>Up to 5.5 mg/kg (below a pondlike structure) Goerlitz (1992)</p>	
<p>A U.S. Navy facility (Kitsap County, WA)</p> <p>- was active from 1966-1970; survey taken in 1974.</p>					<p>&lt; limit of detection to 5 mg/L (concentration in water table below the sediments [see next column] Goerlitz (1992)</p>	<p>&lt;limit of detection to 5.5 mg/kg (below a pond-like structure used to trap washwater from the facility where projectiles were cleaned. Goerlitz (1992)</p>	
Unidentified munitions disposal site					<p>1-47 µg/L (detected in 7 of 7 GW wells) Richards and Junk (1986), cited in Talmage et al. (1999)</p>		
Unspecified AAP					<p>70 µg/L (GW from a water supply well) Jenkins et al. (1986)</p>		

	Amount discharged from facility	Concentration in untreated wastewaters	Soil	Surface Water	Ground Water	Sediments	Comments
Nebraska Ordinance Works			0.5 - 1247 mg/kg (median, 19.5 mg/kg) (10/40 samples) Walsh and Jenkins (1992)				
Former Nebraska Ordinance Plant, Mead, NE			Below detection limit (0.979)-4,460 mg/kg Price et al. (1997)				
Newport IN			0.5 - 12,203 mg/kg (median, 38.6 m/kg) (11/11 samples) Walsh and Jenkins (1992)				
Weldon Springs Training Area			0.5 mg/kg (1/29 samples) Walsh and Jenkins (1992)				
Raritan Arsenal, NJ			0.5-4.38 (mean, 2.4 mg/kg) (2/22 samples) Walsh and Jenkins (1992)				

	Amount discharged from facility	Concentration in untreated wastewaters	Soil	Surface Water	Ground Water	Sediments	Comments
Hawthorne AAP			2.6-8112 mg/kg (median, 127 mg/kg) (5/8 samples) Walsh and Jenkins (1992)	No surface water exists on the site (USA CHPPM 2005).	<0.5(detection limit) - 2600 µg/L Walsh and Jenkins (1992)		USA CHPPM (2005) says RDX was a chemical of concern in soil and groundwater in three areas on site. Soil and groundwater exposure concentrations used in the risk assessment for these areas were 11.24 to >60,000 mg/kg (soil) and 2,600 to µg/L (groundwater in two areas; third area had no RDX in groundwater).
Hastings East Park			0.5 mg/kg (1/24 samples) Walsh and Jenkins (1992)				
Eagles River Flats (AL) (Explosive Ordnance Disposal site and impact area)			0.044 – 0.076 mg/kg (4/216 samples) Walsh and Jenkins (1992)				
Camp Shelby (MO) (Explosive Ordnance Disposal site and impact areas)			0.5-3.83 mg/kg (mean, 2.2 mg/kg) (2/7 samples) Walsh and Jenkins (1992)				

	Amount discharged from facility	Concentration in untreated wastewaters	Soil	Surface Water	Ground Water	Sediments	Comments
Joliet AAP			0.1-3574 mg/kg; detection limit was 0.1 mg/kg; 13 or 40 samples exceeded the detection limit Simini et al. (1995)				
Umatilla Army Depot (AD), OR			3000 mg/kg Funk et al. (1993)  RDX not detected in 11 samples Walsh and Jenkins (1992)				
Volunteer AAP							According to USA CHPPM (2005) RDX was identified as chemical of potential concern in surface water of a drainage basin and did not result in a risk driver.
Naval Surface Warfare Center			<detection limit to 3.3 mg/kg (dry weight) Grant et al. (1995)				



	Amount discharged from facility	Concentration in untreated wastewaters	Soil	Surface Water	Ground Water	Sediments	Comments
Survey or open burning and open detonation sites at several AAPs and depots: Holston, Iowa, Kansas, Louisiana, Ravenna, Fort Wingate, and Milan AAPs and the Picatinny Arsenal			1200-74000 mg/kg (highest conc. in the residue and soil surface); concentrations in residues and soil (surface to 18 in depth) Newell (1984)				
Fort Polk, Louisiana, a low-order detonation event			0.1 to 16 mg/kg Pennington et al. (2005)				
Fort Polk, Louisiana, around a tank target			0.04 to 2,390 mg/kg (for 100 discrete samples) Pennington et al. (2005)				
Camp Edwards, Massachusetts Military Reservation (MMR) near Falmouth, MA (Data from 1 October 2003 through 30 August 2004)			min. 14 µg/kg max. 15000.0 µg/kg mean 1121.08 µg/kg  # of detects (26), # of samples (711) Pennington et al. (2005)		min. 0.25 µg/L max. 220.0 µg/L mean 6.33 µg/L  # of detects (415), # of samples (1898) Pennington et al. (2005)		

	Amount discharged from facility	Concentration in untreated wastewaters	Soil	Surface Water	Ground Water	Sediments	Comments
Raritan Arsenal (New Jersey)			0.4-4.38 mg/kg; median 2.44 µg/kg (2/22 samples) Walsh and Jenkins (1992)				
VIGO Chemical Plant (Indiana)			RDX not detected in 2 samples Walsh and Jenkins (1992)				
Chickasaw Ordnance Works, TN			RDX not detected in 2 samples Walsh and Jenkins (1992)				
Sangamon Ordnance Plant (Illinois)			RDX not detected in 2 samples Walsh and Jenkins (1992)				
Lexington-Blueglass Depot (KY)			RDX not detected in 13 samples Walsh and Jenkins (1992)				

\*Empty cells indicate concentrations were not reported.

AAP: Army Ammunition Plant

BRAC: U.S. Department of Defense (DOD) Base Realignment and Closure

## References

- Bender, E.S., Robinson, P.F., Moore, M.W., Thornton, W.D., Asaki, A.E. 1977. Preliminary environmental survey of Holston Army Ammunition Plant, Kingsport, TN. AD-A043 662. U.S. Army Chemical Systems Laboratory, Aberdeen Proving Ground, MD. [Cited in Talmage et al. 1999]
- Best, E.P.H., Zappi, M.E., Fredrickson, H.L., Sprecher, S.I., Larson, S.L., Miller, J.L. 1997. Screening of aquatic and wetland plant species for phytoremediation of explosives-contaminated groundwater from the Iowa Army Ammunition Plant. Technical Report EL-97-2. U.S. Army Corps of Engineers, Waterways Experiment Station.
- Burrows, E.P., Rosenblatt, D.H., Mitchell, W.R., Parmer, D.L. 1989. Organic explosives and related compounds: environmental and health considerations. Ad-A210 554. U.S. Army Biomedical Research and Development Laboratory, Fort Detrick, MD. [Cited in Talmage et al. 1999]
- Envirodyne Engineers, Inc. 1980. Milan Army Ammunition Plant contamination survey. Final report. AD-BO53362. Envirodyne Engineers, Inc., St. Louis, MO. [Cited in Talmage et al. 1999]
- Funk, S.B., Roberts, D.J., Crawford, D.L., Crawford, R.L. 1993. Initial-phase optimization for bioremediation of munition compound-contaminated soils. Appl Environ Microbiol 59:2171-2177. [Cited in Talmage et al. 1999]
- Goerlitz, D.F. 1992. A review of studies of contaminated groundwater conducted by the U.S. Geological Survey Organics Project, Menlo Park, California, 1961-1990. Environ Sci Pollut Control Ser 4:295-355. [Cited in Talmage et al. 1999]
- Grant, C.L., Jenkins, T.F., Myers, K.F., McCormick, E.F. 1995. Holding-time estimates for soils containing explosives residues: comparison of fortification vs. field contamination. Environ Toxicol Chem 14:1865-1874. [Cited in Talmage et al. 1999]
- Gregory, R.G., Elliott, W.G. 1987. Remedial investigation at Louisiana Army Ammunition Plant. Final Report. AMXTH-IR-CR-87110. Environmental and Science Engineering, Inc. Gainesville, Source is in a foreign language. Cannot confirm. U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, MD. [Cited in Talmage et al. 1999]
- Jenkins, T.F., Leggett, D.C., Grant, C.L., Bauer, C.F. 1986. Reversed-phase high-performance liquid chromatographic determination of nitroorganics in munitions wastewater. Anal Chem 58:170-175. [Cited in Talmage et al. 1999]

- Jenkins, T.F., Grant, C.L. 1987. Comparison of extraction techniques for munitions residues in soil. *Anal Chem* 59:1326-1331. [Cited in Talmage et al. 1999]
- Newell, E.L., JR. 1984. Phase 3. Hazardous waste study NO. 37-26-0147-84. Summary of AMC open burning/open detonation ground evaluations, November 1981-September 1983. Department of the Army, U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD. [Cited in Talmage et al. 1999]
- Pennington, J.C., Jenkins, T.F., Thiboutot, S., Ampleman, G., Clausen, J., Hewitt, A.D., Lewis, J., Silverblatt, B., Marois, A., Gagnon, A., Brousseau, P., Zuflet, J.E., Poe, K., Bouchard, M., Martel, R., Walker, D.D., Ramsey, C.A., Hayes, C.A., Yost, S.L., Bjella, K.L., Trepanier, L., Berry, T.E., Lambert, D.J., Dube, P., Perron, N.M. 2005. Distribution and fate of energetics on DoD test and training ranges: Interim Report 5. Strategic Environmental Research and Development Program. U.S. Army Corps of Engineers, Cold Regions Research & Engineering Laboratory, Hanover, NH.
- Phung, H.T., Bulot, M.W. 1981. Subsurface investigation of metal sludge and explosive disposal pond areas. In: Conway RA, Malloy DC (eds) *Hazardous Solid Waste Testing, First Conference*. ASTM STP 760. American Society for Testing and Materials, Philadelphia, PA. [Cited in Talmage et al. 1999]
- Price, R.A., Pennington, J.C., Larson, S.L., Neumann, D., Hayes, C.A. 1997. Plant uptake of explosives from contaminated soil and irrigation water at the former Nebraska Ordnance Plant, Mead, Nebraska. U.S. Army Corps of Engineers, Waterways Experiment Station. Technical report EL-97-11.
- Richards, J.J., Junk, G.A. 1986. Determination of munitions in water using macroreticular resins. *Anal Chem* 58:723-725. [Cited in Talmage et al. 1999]
- Rosenblatt, D.H. 1986. Contaminated soil cleanup objectives for Cornhusker Army Ammunition Plant. Tech Rep 8603. U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, MD. [Cited in Talmage et al. 1999]
- Simini, M., Wentsel, R.S., Checkai, R.T., Phillips, C.T., Chester, N.A., Major, M.A., Amos, J.C. 1995. Evaluation of soil toxicity at Joliet Army Ammunition Plant, *Environ Toxicol Chem* 14:623-630.
- Small, M.J., Rosenblatt, D.H. 1974. Munitions production products of potential concern as waterborne pollutants: Phase II. AD-919031. U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, MD. [Cited in Talmage et al. 1999]

- Spalding, R.F., Fulton, J.W. 1988. Groundwater munition residues and nitrate near Grand Island, Nebraska, U.S.A. *J Contam Hydrol* 2:139-153 [Cited in Talmage et al. 1999]
- Spanggord, R.J., Gibson, B.W., Keck, R.G., Newell, G.W. 1978. Mammalian toxicological evaluation of TNT wastewaters (“pink water”). Vol. I. Chemistry studies, draft report. AD A059434. US Army Medical Research and Development Command, Fort Detrick, MD. [Cited in Talmage et al. 1999]
- Spanggord, R.J., Mabey, W.R., Mill, T., Chou, T.W., Smith, J.H., Lee, S. 1980. Environmental fate studies on certain munition wastewater constituents. Final report. Phase II: Laboratory studies. SRI International, Menlo Park, CA. AD099256. [US Army Medical Research and Development Command, Fort Detrick, MD. [Cited as Spangoord et al. 1980b in Talmage et al. 1999]
- Spanggord, R.J., Mabey, W.R., Chou, T.W., Lee, S., Alferness, P.L., Tee, D.S., Mill, T. 1983. Environmental fate studies of HMX. Phase II, detailed studies. Final report. SRI International, Menlo Park, CA. [Cited in Talmage et al. 1999]
- Stillwell, J.M., Fischer, M.A., Margard, W.L., Matthews, M.C., Sherwood, B.E., Stanford, T.B. 1977. Toxicological investigations of pilot treatment plant wastewaters at Holston Army Ammunition Plant. Final report, AD A042601. Battelle Columbus Laboratories, Columbus, OH. [Cited in Talmage et al. 1999]
- Talmage, S.S., Opresko, D.M., Maxwell, C.J., Christopher, J.E., Welsh, J.E., Cretella, F.M., Reno, P.H., Daniel, F.B. 1999. Nitroaromatic munition compound: environmental effects and screening values. *Rev Environ Contam Toxicol* 161:1-156.
- Todd, Q., Finger, F., Tuner, R., Morley, D. 1989. Delivery Order 8, Louisiana Army Ammunition Plant: updated remedial investigation. Roy F. Weston, West Chester, PA. U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, MD. [Cited in Talmage et al. 1999]
- Tucker, W.A., Dose, E.V., Gensheimer, G.J. 1985. Evaluation of critical parameters affecting contaminant migration through soils. Final report. U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, MD. [Cited in Talmage et al. 1999]
- US Army CHPPM (U.S. Army Center for Health Promotion and Preventive Medicine). 2005. Relative Source Contribution for RDX. Draft.

Walsh, M.E., Jenkins, T.F. 1992. Identification of TNT transformation products in soil. ADA 225 308. U.S. Army Corps of Engineers, Cold Regions Research & Engineering Laboratory, Hanover, NH.

## **Appendix C**

### **Plant Uptake Studies and Plant Uptake Factors (PUFs)**

Checkai et al. (1996) and Checkai and Simini (1996) conducted site-specific studies to determine the uptake of RDX into edible parts of home garden crops (bush bean, tomato, lettuce, and radish varieties) and field crops (corn, soybean, and alfalfa) grown in uncontaminated (free of explosives) soils at a site that once involved munitions production activities. The plants were irrigated in an environment-controlled greenhouse to water holding capacity throughout the life-cycle of the crop with nominal (actual) irrigation treatments of 2 (1.8), 20 (18), and 100 (90.2) ppb RDX. According to the authors, potential of uptake of RDX by each species was maximized, reduction in uptake due to external environmental stress was minimized, and potential treatment losses due to runoff and drainage were eliminated, thereby maximizing, and making more quantifiable, the impact of RDX treatments. Edible plant tissues selected for analysis included root, stem, leaf, seed, and fruit, and plant uptake factors (PUFs) for irrigation water-to-plant fresh weight were determined. Cataldo et al. (1990) examined uptake of radiolabeled RDX into roots, stems, leaves, pods and stems of bean plants grown for 60 days in several soil types amended with 10 ppm (mg/kg) <sup>14</sup>C-RDX. In addition, these authors also investigated the uptake of RDX into the same plant tissues of beans grown in hydroponic solutions that were amended with 1-25 mg/L (ppm) <sup>14</sup>C-RDX. Lachance et al. (2003) investigated bioaccumulation of non-radiolabeled RDX in alfalfa after 16-day exposure in fresh or weathered/aged natural sandy loam soil amended with 10,000 mg RDX/kg soil (measured: 9740 mg/kg for freshly amended soil and 9537 mg/kg for weathered/aged soil). The bioconcentration factor (defined as mg/kg dry mass plant compartment divided by the measured concentration mg/kg dry soil) reported for alfalfa was 0.27 and 0.66 in freshly amended and weathered/aged soil, respectively. The same authors also exposed alfalfa for 42 days to radiolabeled RDX at nominal concentrations of 100 and 1000 mg/kg in the sandy loam soil (measured concentrations were 87 and 998 mg/kg, respectively) and reported bioconcentration factors of 79 and 6.8, respectively.

Harvey et al. (1991) studied plant uptake of RDX into selected agronomic species, bush beans and wheat in 1- and 7-day hydroponic solutions amended with 10 ppm (mg/kg) RDX containing radiolabeled RDX. Uptake of RDX into roots, stems, and leaves was determined. Fellows et al. (1995) investigated the uptake of RDX in four species of crop plants (corn, alfalfa, spinach, and carrot) grown from seed to maturity (70 to 90 days) in a low-fertility desert soil amended with 15 µg/g RDX. Uptake of RDX was determined in shoot, root, and seed/blossom of spinach and alfalfa, shoot and root of carrots, and tassel, leaves, stem, ear, and root of corn. Larson et al. (1999) studied the uptake of RDX into tomato, radish, and tassels and leaves of corn grown in soil irrigated with 1.0 µg/ml RDX. Price et al. (1997) conducted greenhouse studies in which selected agronomic species, corn, tomato, lettuce, and radish were used to measure plant uptake of RDX into usable (edible) plant tissues from soil contaminated with 0.58 to 580 mg/kg RDX or irrigation water contaminated with 100 or 1,000 µg/L RDX.

Table C-1 presents a summary of uptake factors (bioconcentration ratios) for edible species of field and garden crop plants grown in RDX-amended soil. Similarly, Table C-2 presents uptake factors for plants irrigated with water containing RDX. From Table C-1, the mean soil-to-plant bioconcentration ratio for RDX is 5.81, with a median and geometric mean of 2.39 and 1.60, respectively. From Table C-1, the mean water-to-plant bioconcentration ratio is 0.91, with

median and geometric mean of 0.3 and 0.44, respectively. The mean value for all results (i.e., combining soil-to-plant and water-to-plant bioconcentration ratios from Tables C-1 and C-2) is 4.04; the median is 0.84, and the geometric mean is 1.01.



**Table C-1. Soil-to-Plant Uptake Factors**

<b>Plant type and part</b>	<b>Supply medium</b>	<b>Medium conc. (ppb RDX)</b>	<b>Cited as dm or fm</b>	<b>Soil foc<sup>a</sup></b>	<b>Plant conc. (ppb RDX)</b>	<b>Cited as dm or fm</b>	<b>Plant dm fraction</b>	<b>BCR result<sup>b</sup></b>	<b>Reference</b>
Bean pod	Soil	10,000	dm	0.005	1,292	fm		0.06	Cataldo et al. 1990
Tomato fruit	Soil	50,300	fm	0.030	74,519	dm	0.07	0.10	Price et al. 1997
Alfalfa	Soil	9,740	dm	-	2610	dm	-	0.27 <sup>d</sup>	Lachance et al. 2003
Tomato fruit	Soil	1,640	fm	0.030	7,867	dm	0.07	0.30	Price et al. 1997
Tomato fruit	Soil	1,640	fm	0.030	9,719	dm	0.07	0.40	Price et al. 1997
Alfalfa	Soil	9,537	dm	-	6321	dm	-	0.66 <sup>d</sup>	<b>Lachance et al. 2003</b>
Tomato fruit	Soil	7,675	fm	0.030	79,595	dm	0.07	0.70	Price et al. 1997
Lettuce leaves	Soil	1,770	fm	0.030	10,000	dm	0.05	0.75	Price et al. 1997
Lettuce leaves	Soil	1,640	fm	0.030	9,620	dm	0.05	0.77	Price et al. 1997
Lettuce leaves	Soil	673	fm	0.030	7,900	dm	0.05	1.55	Price et al. 1997
Bush seed	Soil	10,000	dm	0.017	13,535	fm		2.23	Cataldo et al. 1990
Lettuce leaves	Soil	5,800	fm	0.051	62,480	dm	0.05	2.39	Price et al. 1997
Lettuce leaves	Soil	5,800	fm	0.054	62,480	dm	0.05	2.53	Price et al. 1997
Lettuce leaves	Soil	7,675	fm	0.030	154,000	dm	0.05	2.65	Price et al. 1997

Plant type and part	Supply medium	Medium conc. (ppb RDX)	Cited as dm or fm	Soil foc <sup>a</sup>	Plant conc. (ppb RDX)	Cited as dm or fm	Plant dm fraction	BCR result <sup>b</sup>	Reference
Bush seed	Soil	10,000	dm	0.072	4,025	fm		2.90	Cataldo et al. 1990
Lettuce leaves	Soil	50,300	fm	0.030	1,172,000	dm	0.05	3.07	Price et al. 1997
Lettuce leaves	Soil	5,800	fm	0.030	154,000	dm	0.05	3.50	Price et al. 1997
Spinach shoot	Soil	15,000	dm	0.005	832,000	dm	0.20	5.55	Fellows et al. 1995
Lettuce leaves	Soil	5,800	fm	0.064	11,750	dm	0.05	5.58	Price et al. 1997
Lettuce leaves	Soil	5,800	fm	0.067	117,960	dm	0.05	5.88	Price et al. 1997
Lettuce leaves	Soil	5,800	fm	0.019	405,000	dm	0.05	5.99	Price et al. 1997
Alfalfa	Soil	998	dm	-	6820	dm	-	6.8 <sup>d</sup>	Lachance et al. 2003
Alfalfa	Soil	87	dm	-	6871	dm	-	79 <sup>d</sup>	Lachance et al. 2003
<b>Mean</b>								<b>5.81</b>	
<b>Median</b>								<b>2.39</b>	
<b>Geometric Mean</b>								<b>1.60</b>	
<b>Maximum</b>								<b>79</b>	

<sup>a</sup>Fraction of organic carbon (see McKone and Maddalena 2007)

<sup>b</sup>Reported in McKone and Maddalena (2007)

<sup>d</sup>Not reported in McKone and Maddalena (2007)

**Table C-2. RDX Uptake into Plant from RDX-Amended Water**

<b>Plant type and part</b>	<b>Supply medium</b>	<b>Medium conc. (ppb RDX)</b>	<b>Cited as dm or fm</b>	<b>Plant conc. (ppb RDX)</b>	<b>Cited as dm or fm</b>	<b>Plant dm fraction</b>	<b>BCR result</b>	<b>Reference</b>
Tomato fruit	water	100	fm	16	fm		0.16	Checkai and Simini 1996; Checkai et al. 1996
Tomato fruit	water	20	fm	6	fm		0.30	Checkai and Simini 1996; Checkai et al. 1996
Tomato fruit	water	2	fm	11	fm		5.50	Checkai and Simini 1996; Checkai et al. 1996
Lettuce leaves	water	134	fm	1,600	dm	0.03	0.30	Price et al. 1997
Lettuce leaves	water	100	fm	77	fm		0.77	Checkai and Simini 1996; Checkai et al. 1996
Lettuce leaves	water	20	fm	18	fm		0.90	Checkai and Simini 1996; Checkai et al. 1996
Lettuce leaves	water	812	fm	21,320	dm	0.05	1.31	Price et al. 1997
Lettuce leaves	water	406	fm	11,140	dm	0.05	1.37	Price et al. 1997
Bush bean fruit	Water	100	fm	7	fm		0.07	Checkai and Simini 1996; Checkai et al. 1996
Bush bean fruit	Water	20	fm	4	fm		0.20	Checkai and Simini 1996; Checkai et al. 1996
Radish root	Water	100	fm	14	fm		0.14	Checkai and Simini 1996; Checkai et al. 1996
Radish root	Water	20	fm	6	fm		0.3	Checkai and Simini 1996; Checkai et al. 1996

<b>Plant type and part</b>	<b>Supply medium</b>	<b>Medium conc. (ppb RDX)</b>	<b>Cited as dm or fm</b>	<b>Plant conc. (ppb RDX)</b>	<b>Cited as dm or fm</b>	<b>Plant dm fraction</b>	<b>BCR result</b>	<b>Reference</b>
Radish root	Water	2	fm	9	fm		0.45	Checkai and Simini 1996; Checkai et al. 1996
<b>Mean</b>							<b>0.91</b>	
<b>Median</b>							<b>0.3</b>	
<b>Geometric Mean</b>							<b>0.44</b>	
<b>Maximum</b>							<b>5.5</b>	

## References

- Cataldo, D.A., Harvey, S.D., Fellows, R.J. 1990. An evaluation of the environmental fate and behavior of munitions material (TNT, RDX) in soil and plant systems. Environmental fate and behavior of RDX. PNL-7529. Prepared for the U.S. Army Biomedical Research and Development Laboratory, Fort Detrick, Fredrick MD.
- Checkai, R.T., Simini, M. 1996. Phytophysical response of crops to irrigation waters containing low concentrations of RDX and TNT: Ecotoxicological implications. US. Army Edgewood Research. Geo-Centers, Inc.
- Checkai, R.T., Simini, M., Harvey, S.D. 1996. Plant Uptake of RDX and TNT Utilizing Site Specific Criteria for the Cornhusker Army Ammunition Plant (CAAP), Nebraska. US Army ERDEC Technical Report. Project Order No 560786M8AA.
- Fellows RJ, Harvey SD, Cataldo DA. 1995. Evaluation of the metabolic fate of munitions material (TNT, RDX) in plant systems and initial assessment of material interaction with plant genetic material: Validation of the metabolic fate of munitions material (TNT, RDX) in mature crops. PNL-10825/UC-402. Pacific Northwest Laboratories, Richland, WA, USA. [Cited in McKone and Maddalena, 2007].
- Fellows, R.J., Driver, C.R., Cataldo, D.A., Harvey, S.D. 2006. Bioavailability of hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) to the prairie vole (*Microtus ochrogaster*). Environmental Toxicology and Chemistry 25(7): 1881-1886.
- Harvey, S.D., Fellows, R.J., Cataldo, D.A., Bean, R.M. 1991. Fate of the Explosive Hexahydro-1,3,5- Trinitro-1,3,5-Triazine (RDX) in Soil and Bioaccumulation in Bush Bean Hydroponic Plants. Env. Toxic. & Chem. 10: 845- 855.
- Lachance, B., Rocheleau, S., Hawari, J., Gong, P., Leduc, F., Apte, J., Sarrazin, M., Martel, M., Bardai, G., Dodard, S., Sunahara, G.I. 2003. Bioaccumulation of nitro-heterocyclic and nitroaromatic energetic materials in terrestrial receptors in a natural sandy loam soil. Biotechnology Research Institute. National Research Council Canada.
- Larson, S.L., Jones, R.P., Escalon, L., Parker, D. 1999. Classification of explosives transformation products in plant tissue. Environmental Toxicology and Chemistry 18(6): 1270-1276.
- McKone, T.E., Maddalena, R.L. 2007. Plant uptake of organic pollutants from soil: Bioconcentration estimates based on models and experiments. Environmental Toxicology and Chemistry 26(12): 2494-2504.
- Price, R.A., Pennington, J.C., Neumann, D., Hayes, C.A., Larson, S.L. 1997. Plant Uptake of Explosives from Contaminated Soil and Irrigation Water at the Former Nebraska Ordnance Plant, Mead, Nebraska. Technical Report EL-97-11, U.S. Army Engineer Waterways Experiment Station, Vicksburg, MS.

## Appendix D

### Wildlife and Fish Bioaccumulation Studies

Whaley and Leach (1994) investigated the potential of contamination of white-tailed deer harvested during a hunting season from a site used in the past for munition production, storage, and demilitarization and from an off-post site (as a control). Samples of liver and muscle were analyzed for several chemicals, including explosives and several breakdown products of these materials. The investigators found no contamination with RDX in the deer tissues. Deer and white-footed mice were also sampled from Aberdeen Proving Ground, MD, in 1994 and 1995, and white-footed mice from Volunteer Army Ammunition Plant in 1996 (USACHPPM, 2002). No RDX was found in these species, indicating wildlife species inhabiting these areas are not likely to bioaccumulate RDX in their tissues.

Bentley et al. (1977) reported RDX bioaccumulation in the edible portions (muscle) and viscera of bluegill sunfish, channel catfish, and fathead minnows exposed continuously for 28 days to radiolabeled RDX in an intermittent-flow at RDX mean concentrations 0.014 and 1.0 mg/L. Bioaccumulation factors (BAF) values – based on tracking a radioactive label and thus corresponding to molar sum of RDX and all transformation products – were higher at the lower concentration compared to the higher concentration for each species and were slightly higher in the viscera than in the edible portions (muscle) of these species. BAF values ranged from 2.9 to 5.9 in the muscle and from 3.3 to 11 in the viscera. In the muscle, BAF values ranged from 4.0 in the catfish to 5.9 in the fathead minnow at the lower concentration, compared to the range of 2.9 (catfish) to 4.0 (fathead minnow) at the higher concentration. The corresponding values in the viscera ranged from 5.0 (catfish) to 11.0 (fathead minnow) at the lower concentration, compared to the range of 3.3 (catfish) to 8.8 (fathead minnow) at the higher concentration. The Bentley et al. (1977) study indicates lower BAFs for catfish compared to those in the fathead minnow, irrespective of the RDX concentration or tissue. The mean BAF values for muscle at the two tested concentrations were 3.4, 4.1, and 5.0 for the channel catfish, bluegill sunfish, and fathead minnows, respectively. Liu et al. (1983) reported lower BAF values – also based on tracking a radioactive label – of 1.9 (muscle) and 3.1 (viscera) in a 96-hour static test. Belden et al. (2005) investigated RDX accumulation in juvenile catfish following aqueous exposure only (2 mg/L), dietary exposure only (to prey, *L. variegates*, exposed to 10 mg/L of RDX for 4 h), and a combination of dietary and aqueous exposure. The investigators reported BCF value of approximately 2.0 mg/L for the catfish and concluded that dietary exposure to RDX-laden prey is likely to result in little additional accumulation in catfish inhabiting RDX-contaminated sites, indicating that RDX uptake via the aqueous route is the expected dominant uptake pathway while dietary uptake contributes minimally to the overall body burden in fish inhabiting RDX-contaminated sites. Based on the log  $K_{ow}$  of RDX, BAF values ranging from 1.5 to 2.7 mg/L have been estimated for small fish or fish (Belden et al., 2005; Layton et al., 1987; Burrows et al., 1989).

Using the BAFs from the studies in the table below (without the marine mussel data), the average value for all results is 3.6 mL/g, the geometric mean is 3.3 mL/g, and the maximum factor is 5.9 mL/g.

**Table D-1 RDX Bioaccumulation Factors (BAFs) in Fish and Marine Mussel**

Fish	Bioaccumulation Factor (mL/g)		
	Muscle	Viscera	Reference
Channel catfish	4.0 (L) 2.9 (H)	5.0 (L) 3.3 (H)	Bentley et al. (1977)
Bluegill sunfish	4.7 (L) 3.5 (H)	9.0 (L) 6.0 (H)	Bentley et al. (1977),
Fathead minnows	5.9 (L) 4.0 (H)	11.0 (L) 8.8 (H)	Bentley et al. (1977)
Juvenile sheepshead minnows	1.7		Lotufo and Lydy (2005)
Bluegill sunfish	1.9	3.1	Liu et al. (1983)
Juvenile fathead minnows	0.010 g/g	-	Houston and Lotufo (2005)
Juvenile catfish	2.0 (kinetic) ~2.0 (steady state)	-	Belden et al. (2005)
Small fish	2.7 (based on log Kow and bioconcentration equation)	-	Belden et al. (2005)
Fish	2 (calculated from log Kow)		Layton et al. (1987)
Fish	1.5 (calculated from log Kow)	-	Burrows et al. (1989)
Marine mussel	0.77 <sup>a</sup> 0.67 <sup>b</sup> 0.69 <sup>c</sup>	-	Rosen and Lotufo (2007)
Average*	<b>3.6</b>		
Geometric mean*	<b>3.3</b>		
90 <sup>th</sup> percentile	<b>5.0</b>		
Maximum*	<b>5.9</b>		

L = test water mean measured concentration of 0.014 mg/L

H = test water mean measured concentration of 1.0 mg/L

<sup>a</sup>test water nominal concentration of 3 mg/L and measured concentration of 2.79 mg/L

<sup>b</sup>test water nominal concentration of 10 mg/L and measured concentration of 9.12 mg/L

<sup>c</sup>test water nominal concentration of 30 mg/L and measured concentration of 28.4 mg/L

\*Values calculated using experimental values only, that is, excluding calculations based on kinetic/steady state and data from marine mussel

## References

- Belden, J.B., Lotufo, G.R., Lydy, M.J. 2005. Accumulation of Hexahydro-1,3,5-trinitro-1,3,5-triazine in Channel Catfish (*Ictalurus punctatus*) and Aquatic Oligochaetes (*Lumbriculus variegates*). *Environmental Toxicology and Chemistry* 24(8):1962-1967.
- Bentley, R.E., Dean, J.W., Ellis, S.J., Hollister, T.A., LeBlanc, G.A., Sauter, S., Sleight, B.H. 1977. Laboratory evaluation of the toxicity of cyclotrimethylene trinitrate (RDX) to aquatic organisms. AD A061730 . Final report. EG&G Bionomics, Wareham, MA, for U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, MD.
- Brennan, L.A. 1999. Northern bobwhite (*Colinus virginianus*). In Poole A, Gill F, eds. *The Birds of North America*, No. 397. The Birds of North America, Philadelphia, PA, USA, 1-27.
- Burrow, E.P., Rosenblatt, D.H., Mitchell, W.R., Parmer, D.L. 1989. Organic explosives and related compounds; environmental and health considerations. AD-A210 554. U.S Army Biomedical Research and Development Laboratory, Fort Detrick, MD.
- Gogal, R.M., Johnson, M.S., Larsen, C.T., Prater, M.R., Duncan, R.B., Ward, D.L., Lee, R.B., Salice, C.J., Jortner, B., Holladay, S.D. 2003. Dietary Oral Exposure To 1,3,5-Trinitro-1,3,5-Triazine In The Northern Bobwhite (*Colinus virginianus*). *Environmental Toxicology and Chemistry* 22(2):381-387.
- Houston, J.G., Lotufo, G.R. 2005. Dietary Exposure of Fathead Minnows to the Explosives TNT and RDX and to the Pesticide DDT using Contaminated Invertebrates. *Int. J. Environ. Res. Public Health*, 2(2), 286–292.
- Layton, D., Mallon, B., Mitchell, W., Hall, L., Fish, R., Perry, L., Snyder, G., Bogen, K., Malloch, W., Ham, C., Dowd, P. 1987. Conventional weapons demilitarization: a health and environmental effects data base assessment. Explosives and their co-contaminants. Final report, phase II. AD-A220588. Lawrence Livermore National Laboratory, Livermore, CA. U.S. Army Medical Research and Development Command, Frederick, MD.
- Liu, D.H., Bailey, H.C., Pearson, J.G. 1983. Toxicity of a complex munitions wastewater to aquatic organisms. In: Bishop WE, Cardwell RD, Heidolph BB (eds) *Aquatic Toxicology and Hazard Assessment: Sixth Symposium*. ASTM STP 802. American Society for Testing and Materials, Philadelphia, PA, pp 135-150.
- Lotufo, G.R., Lydy, M.J. 2005. Comparative Toxicokinetics of Explosive Compounds in Sheepshead Minnows. *Arch. Environ. Contam. Toxicol.* 49:206-214.
- Rosen, G., Lotufo, G.R. 2007. Toxicity of explosive compounds to the marine mussel, *Mytilus galloprovincialis*, in aqueous exposures. *Ecotoxicol Environ Safety* 68:228-236.
- Talmage, S.S., Opresko, D.M., Maxwell, C.J., et al. 1999. Nitroaromatic Munition Compounds: Environmental Effects and Screening Values. *Rev Environ. Contam. Toxicol.* 161:1-156.



USACHPPM (U.S. Army Center for Health Promotion and Preventive Medicine). 2002. Bioconcentration, bioaccumulation, and biomagnification of nitroaromatic and nitramine explosives and their breakdown products. Toxicology Report No. 87-MA-4677-01. U.S. Army Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, MD.

Whaley, J.E., and Leach, G. 1994. Health risk assessment for consumption of deer muscle and liver from Joliet Army Ammunition Plant. Joliet, Illinois. Final Report. Project No. 75-51-YF23. US Army Environmental Hygiene Agency. Aberdeen Proving Ground, Maryland.

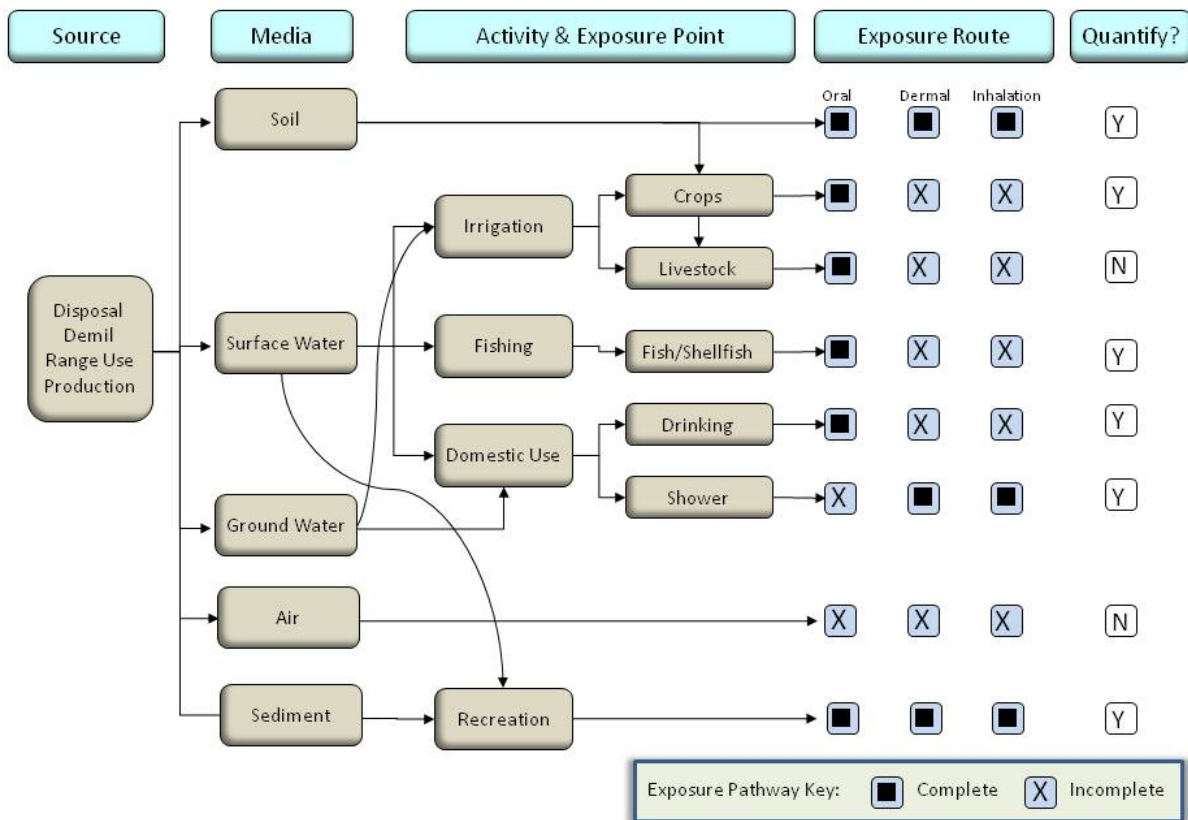
## Appendix E

### Exposure Pathways for the General Population to RDX

Sources of RDX in the environment:

- Industrial production (i.e., a military production facility)
- Military ordnance range use
- Demilitarization operations
- Civilian blasting/shaped charges

Figure E-1. RDX Conceptual Exposure Model



**Table E-1. Potential Exposure Pathways for RDX.**

Environmental Media	Potential Exposure Pathways
Soil	<ul style="list-style-type: none"> <li>• Direct contact with the skin</li> <li>• Incidental ingestion of soil</li> <li>• Inhalation of soil particles*</li> <li>• Ingestion of food plants and livestock* grown with contaminated soils</li> </ul>
Surface Water	<ul style="list-style-type: none"> <li>• Direct contact with the skin</li> <li>• Ingestion of water</li> <li>• Inhalation through showering*</li> <li>• Dermal contact through bathing</li> <li>• Ingestion of fish from contaminated waters</li> <li>• Ingestion of food plants and livestock* irrigated with contaminated water</li> <li>• Ingestion of water from showering*</li> </ul>
Ground Water	<ul style="list-style-type: none"> <li>• Direct contact with the skin</li> <li>• Ingestion of water</li> <li>• Inhalation through showering*</li> <li>• Dermal contact through bathing</li> <li>• Ingestion of food plants and livestock* irrigated with contaminated water</li> <li>• Ingestion of water from showering*</li> </ul>
Air	<ul style="list-style-type: none"> <li>• Inhalation of particulates*</li> </ul>
Sediment	<ul style="list-style-type: none"> <li>• Direct contact with the skin during recreation activities*</li> <li>• Incidental ingestion of sediment during recreation activities*</li> <li>• Inhalation of particles during recreation activities*</li> </ul>

\* indicates those pathways that are minor sources or otherwise not considered in quantification. See main report text and Table E-2 for explanation and discussion.

**Table E-2. Details of potential pathways.**

<b>Media</b>	<b>Secondary Media</b>	<b>Exposure point/location</b>	<b>Exposure route</b>	<b>Receptor</b>	<b>Scenario</b>	<b>Complete?</b>
<b>WATER</b>						
<b>Ground water</b>		<b>Drinking water</b>	<b>Ingestion</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
		<b>Bathing</b>	<b>Dermal</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
		Showering	Inhalation	Child/Adult	Residential	NO. RDX is not volatile
		<b>Showering</b>	<b>Dermal</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
		Showering	Ingestion	Child/Adult	Residential	NO, covered by drinking water
<b>Surface Water</b>		<b>Drinking water</b>	<b>Ingestion</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
		<b>Bathing</b>	<b>Dermal</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
		Showering	Inhalation	Child/Adult	Residential	NO. RDX is not volatile
		Swimming	Dermal	Child/Adult	Recreation	NO, trivial source
		Swimming	Ingestion	Child/Adult	Recreation	NO, trivial source
		Swimming	Inhalation	Child/Adult	Recreation	NO, trivial source and RDX is not volatile
		Showering	Ingestion	Child/Adult	Residential	NO, covered by drinking water
		<b>Fish</b>	<b>Ingestion</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
<b>FOOD</b>						
Store Food		Store Food	Ingestion	Child/Adult	Residential	NO, no data or reason to believe RDX in market foods
<b>Ground water</b>	<b>Irrigation of food crops</b>	<b>Home Grown Produce</b>	<b>Ingestion</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
<b>Surface water</b>		<b>Local Caught Fish</b>	<b>Ingestion</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
Surface water	Game animals drink water	Meat	Ingestion	Child/Adult	Residential	NO, RDX not taken up
Soil	Game animals eat vegetation	Meat	Ingestion	Child/Adult	Residential	NO, RDX not taken up

<b>Media</b>	<b>Secondary Media</b>	<b>Exposure point/location</b>	<b>Exposure route</b>	<b>Receptor</b>	<b>Scenario</b>	<b>Complete?</b>
Soil	Soil clinging to produce	Home Grown Produce	Ingestion	Child/Adult	Residential	NO, trivial amount
Ground Water	Livestock drink water	Meat	Ingestion	Child/Adult	Residential	NO, RDX not taken up
Surface Water	Livestock drink water	Meat	Ingestion	Child/Adult	Residential	NO, RDX not taken up
Soil	Livestock eat vegetation	Meat	Ingestion	Child/Adult	Residential	NO, RDX not taken up
<b>Soil</b>		<b>Crops</b>	<b>Ingestion</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
<b>Soil</b>		<b>Home grown produce</b>	<b>Ingestion</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
<b>AIR</b>						
<b>Indoor Air</b>	<b>All sources</b>	Breathing	Inhalation	Child/Adult	Residential	NO, RDX is not volatile; particulates not expected in indoor environment
<b>Outdoor Air</b>	<b>Industrial production/ Blasting/ Ordinance Use /Demilitarization Operations</b>	Breathing	Inhalation	Child/Adult	Residential	NO, RDX is not volatile; particulates not expected to be transported offsite.
<b>SOIL</b>						
<b>Soil</b>	<b>Incidental hand to mouth</b>		<b>Ingestion</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
	<b>Skin</b>		<b>Dermal</b>	<b>Child/Adult</b>	<b>Residential</b>	<b>YES</b>
	Soil particles		Inhalation	Child/Adult	Residential	NO, particulates not expected to be transported offsite

## Appendix F

### RDX Intake Calculation Spreadsheets

Estimates of chronic daily intakes (CDIs) were calculated from the spreadsheet *RDX intake calculations.xls*. The spreadsheet allows for quick calculation of RDX estimated intakes, given user-provided concentrations of RDX in water, air, or soil. The equations, parameter values, and assumptions used in these spreadsheets are from the U.S. EPA Superfund program and the Risk Assessment Guidance for Superfund (RAGS) documents (e.g., U.S. EPA 1989, 1991a).

The US Department of Energy (DOE), Office of Environmental Management developed a set of online tools, Risk Assessment Information System (RAIS), for use in calculating exposure risk ([http://rais.ornl.gov/prg/for\\_sel\\_data.shtml](http://rais.ornl.gov/prg/for_sel_data.shtml)). This exposure modeling tool, based on EPA's RAGS, allows users to select specific chemicals, land uses, exposure combinations, parameters, and concentrations to estimate site-specific intakes and risks. The assessment focused on potential residential exposures for the general population and did not consider occupational exposure. The equations for residential land can be used to estimate RDX intake from contaminated water ([http://rais.ornl.gov/homepage/tm/for\\_res\\_wa.shtml](http://rais.ornl.gov/homepage/tm/for_res_wa.shtml)) and soil ([http://rais.ornl.gov/homepage/tm/for\\_res\\_so.shtml](http://rais.ornl.gov/homepage/tm/for_res_so.shtml)), in addition to equations that estimate food intakes from agricultural land use ([http://rais.ornl.gov/homepage/tm/for\\_ag.shtml](http://rais.ornl.gov/homepage/tm/for_ag.shtml)). For each potential exposure source (soil, water, or food), these equations represent potential routes and pathways of exposure. For example, there are multiple routes of exposure to contaminated soil (inhalation, ingestion, or dermal contact), so there are multiple equations used to estimate intake. A list of the parameters used in the equations, their values, and references/sources is found in Table F-1 at the end of this appendix.

The exposure modeling spreadsheet includes three tabs – water, soil, and food. Within each tab are the intake equations that correspond to the potential pathways of exposure. For each pathway (e.g., ingestion of water pathway) the chronic daily intake equation is provided, along with a table of default parameter values, which are based on EPA guidance (USEPA, 1991b). Variables such as averaging time and body weight remain constant between CDI equations; but others, such as ingestion rate, may vary depending on the exposure pathway. A complete list of the references used to determine the default values can be found on the RAIS website ([http://rais.ornl.gov/prg/prg\\_ref\\_lu.shtml](http://rais.ornl.gov/prg/prg_ref_lu.shtml) and [http://rais.ornl.gov/homepage/tm/BJCOR271\\_ref.shtml](http://rais.ornl.gov/homepage/tm/BJCOR271_ref.shtml) )

The CDI calculations on the spreadsheet are designed so that users only need to provide the RDX concentration in water or soil, fish bioconcentration factor, or (food) plant concentration in the appropriate cells. The resulting CDI is located in the cells that are highlighted in blue. The calculation table is set up, using default parameters, so that CDIs can be calculated for children, adults, or a person who is exposed through both child- and adulthood. The individual parameter values can be changed if appropriate situation-specific information is available.

**Table F-1 Parameters used to calculate chronic daily intake estimates of RDX.**

Variable		Default or Value	Source	Pertinent Pathways
ABS	Absorption Factor	0.015 (organics)	U.S. EPA 1992	Dermal Contact with Soil
AF	Adherence Factor	0.2 mg/cm <sup>2</sup> (child) 0.07 mg/cm <sup>2</sup> (adult)	RAGs Part E (U.S. EPA, 2004)	Dermal Contact with Soil
AT	Averaging Time	365 days/year ED	U.S. EPA (1989, 1991b)	Ingestion of Water, Soil, and Food; Dermal Contact with Water and Soil
BF	Bioaccumulation Factor, fish	5.0 L/kg	Obtained from the literature, see Appendix D	Ingestion of Fish
BW	Body Weight	15 kg (child) 70 kg (adult)	U.S. EPA (1991a,b)	Ingestion of Water, Soil, and Food; Dermal Contact with Water and Soil
CP	Concentration in Produce	Calculated	Obtained from sample data.	Ingestion of Produce
CPF	Contaminated Plant Fraction	0.25 (residential)	U.S. EPA (1998) p. 6-6	Ingestion of Produce
CS	Concentration in Soil	Calculated – mg/kg	Obtained from sample data (in this example soil concentration equals the SSL – 5.5 mg/kg. (U.S. EPA, 2008)	Ingestion of Soil; Dermal Contact with Soil
CW	Concentration in Water	Calculated – mg/L	Obtained from sample data	Ingestion of Water and Fish; Dermal Contact with Water

Variable		Default or Value	Source	Pertinent Pathways
ED	Exposure Duration	6 years (child) 24 years (adult)	OSWER Directive – (U.S. EPA 1991b)	Ingestion of Water, Soil, and Food; Dermal Contact with Water and Soil
EF	Exposure Frequency	350 days/year	OSWER Directive – (U.S. EPA 1991b)	Ingestion of Water, Soil, and Food; Dermal Contact with Water and Soil
EFf	Fish Consumption Frequency	350 days/year	RAGS ( U.S. EPA 1989)	Ingestion of Fish
ET	Exposure Time (showering)	1 hour/day (child) 0.58 hours/day (adult)	RAGS Part E (U.S. EPA 2004)	Dermal Contact with Water
FI	Fraction Ingested	1 (unitless)	RAGS – (U.S. EPA 1989)	Ingestion of Fish and Beef
IRfi	Ingestion Rate (fish)	0.054 kg/day (child and adult)	OSWER Directive (U.S. EPA 1991b, 1995)	Ingestion of Fish
IRfr	Ingestion Rate (fruit)	0.0148 kg/day (child) 0.0562 kg/day (adult)	U.S. EPA EFH Table 13-61; U.S. EPA (1997) and EPA (1998) (Table C-1-2)	Ingestion of Produce
IRs	Ingestion Rate (soil)	0.0002 kg/day (child) 0.0001 kg/day (adult)	U.S. EPA (1991b)	Ingestion of Soil



Variable		Default or Value	Source	Pertinent Pathways
IRw	Ingestion Rate (water)	1 L/day (child) 2 L/day (adult)	U.S. EPA (1989); OSWER Directive EPA (1991b)  U.S. EPA (2008)	Ingestion of Water
IRv	Ingestion Rate (vegetables)	0.0104 kg/day (child) 0.0285 kg/day (adult)	U.S. EPA EFH (Table 13-61; U.S. EPA 1997)  and U.S. EPA (1998) (Table C-1-2)	Ingestion of Produce
Kp	Permeability Constant	0.000349 cm/hour	Dermal Exposure Assessment (U.S. EPA 1992)	Dermal Contact with Water
SAs	Available Surface Area (soil)	0.28 m <sup>2</sup> /day (child) 0.57 m <sup>2</sup> /day (adult)	Average SA for head, hands, forearms, and lower legs (child includes feet also)  RAGS Part E (U.S. EPA 2004)	Dermal Contact with Soil
SAw	Available Surface Area (water)	1.8 m <sup>2</sup> (adult) 0.66 m <sup>2</sup> (child)	RAGS Part E (U.S. EPA 2004)	Dermal Contact with Water

## References

U.S. EPA (U.S. Environmental Protection Agency). 1988. Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX). IRIS (Integrated Risk Information System). NCEA (National Center for Environmental Assessment). Available at: <http://cfpub.epa.gov/ncea/iris/index.cfm>

U.S. EPA (U.S. Environmental Protection Agency). 1989. [Risk assessment guidance for Superfund, Volume I: Human health evaluation manual \(Part A\)](#). Interim Final. Office of Emergency and Remedial Response. EPA/540/1-89/002.

U.S. EPA (U.S. Environmental Protection Agency). 1991a. Human health evaluation manual, supplemental guidance: "[Standard default exposure factors](#)". OSWER Directive 9285.6-03.

U.S. EPA (U.S. Environmental Protection Agency). 1991b. [Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual \(Part B, Development of Risk-Based Preliminary Remediation Goals\)](#). Office of Emergency and Remedial Response. EPA/540/R-92/003. December 1991

U.S. EPA (U.S. Environmental Protection Agency). 1992. Dermal Exposure Assessment: Principles and Application. Interim Report. Office of Research and Development. EPA/600/8-91/011B.

U.S. EPA (U.S. Environmental Protection Agency). 1995. Supplemental guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment (Interim Guidance). Office of Health Assessment, Waste Management Division.

U.S. EPA (U.S. Environmental Protection Agency). 1996a. [Soil Screening Guidance: User's Guide](#). Office of Emergency and Remedial Response. Washington, DC. OSWER No. 9355.4-23.

U.S. EPA (U.S. Environmental Protection Agency). 1996b. [Soil Screening Guidance: Technical Background Document](#). Office of Emergency and Remedial Response. Washington, DC. OSWER No. 9355.4-17A.

U.S. EPA (U.S. Environmental Protection Agency). 1997. [Exposure Factors Handbook](#). Office of Research and Development, Washington, DC. EPA/600/P-95/002Fa.

U.S. EPA (U.S. Environmental Protection Agency). 1998. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Office of Solid Waste, Washington, DC. EPA530-D-98-001A <http://www.epa.gov/epaoswer/hazwaste/combust/risk.htm>

U.S. EPA (U.S. Environmental Protection Agency). 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual. Office of Superfund Remediation and Technology Innovation, Washington, DC. EPA540-R-99-005.

U.S. EPA (U.S. Environmental Protection Agency). 2008. Regional Screening Levels (RSL) for Chemical Contaminants at Superfund Sites. RSL Table Update.

**Ingestion of Water Pathway**

Receptor	CW mg/L	IR L/day	EF days/year	ED years	BW kg	AT days/year	Chronic Daily Intake (CDI) mg/kg-d
Child		1	350	6	15	2190	0
Adult		2	350	24	70	8760	0
Adult+Child		2	350	30	70	10950	0

$$\text{CDI (mg/kg-d)} = \frac{\text{CW} \times \text{IR} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Variable		Value Used	Explanation/source
AT	Averaging Time	365 days/year x ED	Averaging time for noncarcinogens (EPA 1989a, 1991b)
BW	Body Weight	70 kg (adult)	Adult (EPA 1991b)
		15 kg (child)	
CW	Concentration in Water	chemical specific (mg/L; pCi/L)	
ED	Exposure Duration	24 years (adult)	Residential exposure for a 30-year duration (OSWER Directive, EPA 1991b)
		6 years (child)	
EF	Exposure Frequency	350 days/year	OSWER Directive (EPA 1991b)
IR	Ingestion Rate	2 L/day (adult)	EPA 2009
		1 L/day (child)	

**Dermal Contact with Water While Showering**

Receptor	CW mg/L	EF days/year	ED years	ET hour/.day	SA m <sup>2</sup>	Kp cm/hour	BW kg	AT days/year	Chronic Daily Intake (CDI) mg/kg
Child		350	6	1	0.66	0.00035	15	2190	0
Adult		350	24	0.58	1.8	0.00035	70	8760	0
Adult+Child		350	30	0.58	1.8	0.00035	70	10950	0

$$\text{CDI (mg/kg)} = \frac{\text{CW} \times \text{EF} \times \text{ED} \times \text{ET} \times \text{SA} \times \text{Kp} \times (\text{L}/1000\text{cm}^3) \times (10000\text{cm}^2/\text{m}^2)}{\text{BW} \times \text{AT}}$$

Variable		Value Used	Explanation/source
AT	Averaging Time	365 days/year x ED	Averaging time for noncarcinogens (EPA 1989a, 1991b)
		70 kg	
BW	Body Weight	15 kg	Adult (EPA 1991b)
CW	Concentration in Water	chemical specific (mg/L; pCi/L)	
ED	Exposure Duration	24 years (adult)	Residential exposure for a 30-year duration (OSWER Directive, EPA 1991b)
		6 years (child)	
EF	Exposure Frequency	350 days/year	OSWER Directive (EPA 1991b)
ET	Exposure Time	0.58 hours/day (adult)	RAGs Part E (EPA 2004) and EPA 2009
		1 hr/day (child)	
Kp	Permeability Constant	Chemical specific (cm/hour) (.000349)	Dermal Exposure Assessment (EPA 1992a)
SA	Available Surface Area	1.8 m <sup>2</sup> (adult)	RAGs Part E (EPA 2004)
		0.66 m <sup>2</sup> (child)	

**Ingestion of Soil Pathway**

Receptor	CS mg/kg	IR kg/day	EF days/year	ED years	BW kg	AT days/year	Chronic Daily Intake (CDI) mg/kg-d
Child		0.0002	350	6	15	2190	0
Adult		0.0001	350	24	70	8760	0
Adult+Child		0.0001	350	30	70	10950	0

$$\text{CDI (mg/kg-d)} = \frac{\text{CS} \times \text{IR} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Variable		Value Used	Explanation/source
AT	Averaging time	365 days/year x ED	Averaging time for noncarcinogens (EPA 1989a, 1991b)
BW	Body Weight	70kg (adult)	OSWER Directive, EPA 1991a
		15kg (child)	
CS	Concentration in soil or sediment	Chemical-specific (mg/kg)	
ED	Exposure duration	6 years (child)	Two parts (child and adults) exposure for 30-years of exposure duration (OSWER Directive, EPA 1991b)
		24 years (adult)	
EF	Exposure frequency	350 days/year	OSWER Directive (EPA 1991a, 1991b)
IR	Ingestion Rate	0.0002 kg/day (child)	EPA 1991b
		0.0001 kg/day (adult)	

**Dermal Contact with Soil Pathway**

Receptor	Dermal Contact with Soil Pathway								Chronic Daily Intake (CDI)
	CS	EF	ED	SA	AF	ABS	BW	AT	
	mg/kg	days/year	years	m <sup>2</sup> /day	mg/cm <sup>2</sup>		kg	days/year	mg/kg-d
Child		350	6	0.28	0.2	0.015	15	2190	0
Adult		350	24	0.57	0.07	0.015	70	8760	0
Adult+Child		350	30	0.57	0.07	0.015	70	10950	0

$$CDI \text{ (mg/kg-d)} = \frac{CS \times EF \times ED \times SA \times AF \times ABS \times (kg/1000000mg) \times (10000cm^2/m^2)}{BW \times AT}$$

Variable		Value Used	Explanation/source
ABS	Absorption Factor	0.001 (inorganics) 0.015 (organics)	Equivalent to 0.1% for inorganics and 1.5% for organics (EPA 1992b)
AF	Adherence Factor	0.07 mg/cm <sup>2</sup> (adult)	RAGs Part E (EPA 2004)
		0.2 mg/cm <sup>2</sup> (child)	
AT	Averaging time	365 days/year x ED	Averaging time for noncarcinogens (EPA 1989a, 1991b)
BW	Body Weight	70kg (adult)	OSWER Directive, EPA 1991a
		15kg (child)	
CS	Concentration in soil or sediment	Chemical-specific (mg/kg)	
ED	Exposure duration	6 years (child)	Two parts (child and adults) exposure for 30-years of exposure duration (OSWER Directive, EPA 1991b)
		24 years (adult)	
EF	Exposure Frequency	350 days/year	OSWER Directive (EPA 1991b)
SA	Available Surface Area	0.57 m <sup>2</sup> /day (adult)	Average surface area for head, hands, forearms, and lower legs for an adult. Child includes the adult areas plus feet. (RAGs Part E - EPA 2004)
		0.28 m <sup>2</sup> /day (child)	

**Ingestion of Homegrown Produce Pathway**

Soil to Plant Uptake

Receptor	CP mg/kg	IRf kg/day	IRv kg/day	CPF	EF days/year	ED year	BW kg	AT days/year	Chronic Daily Intake (CDI) mg/kg-day
Child		0.0148	0.0104	0.25	350	6	15	2190	0
Adult		0.0562	0.0285	0.25	350	24	70	8760	0
Adult+Child		0.0562	0.0285	0.25	350	30	70	10950	0

**Ingestion of Homegrown Produce Pathway**

Water to Plant Uptake

Receptor	CP mg/kg	IRf kg/day	IRv kg/day	CPF	EF days/year	ED year	BW kg	AT days/year	Chronic Daily Intake (CDI) mg/kg-day
Child		0.0148	0.0104	0.25	350	6	15	2190	0
Adult		0.0562	0.0285	0.25	350	24	70	8760	0
Adult+Child		0.0562	0.0285	0.25	350	30	70	10950	0

$$\text{CDI (mg/kg-day)} = \frac{\text{CP} \times (\text{IRf} + \text{IRv}) \times \text{CPF} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$



Variable		Value Used	Explanation/source
AT	Averaging Time	365 days/year x ED	Averaging time for noncarcinogens (EPA 1989a, 1991b)
BW	Body Weight	70kg (adult)	OSWER Directive, EPA 1991a
		15kg (child)	OSWER Directive, EPA 1991b
CP	Concentration in produce	chemical-specific (mg/kg)	
CPF	Contaminated plant fraction	0.25 (resident) 1.0 (agriculture)	<a href="#">EPA 1998 pg. 6-6</a>
ED	Exposure Duration	6 years	Two parts (child and adults) exposure for 30-years of exposure duration (OSWER Directive, EPA 1991b)
		24 years	
EF	Exposure frequency	350 days/year	OSWER Directive (EPA 1991a, 1991b)
IRf	Fruit Ingestion Rate	0.0562 kg/day (adult)	EPA EPF (Table 13-61) and EPA 1998 (Table C-1-2)
		0.0148 kg/day (child)	EPA EPF (Table 13-61) and EPA 1998 (Table C-1-2)
IRv	Vegetable Ingestion Rate	0.0285 kg/day (adult)	EPA EPF (Table 13-61) and EPA 1998 (Table C-1-2)
		0.0104 kg/day (child)	EPA EPF (Table 13-61) and EPA 1998 (Table C-1-2)

**Ingestion of Fish Pathway**

Receptor	C <sub>fish</sub> mg/kg	IR <sub>f</sub> kg/day	FI	EF <sub>f</sub> days/year	ED year	BW kg	AT years * day/year	Chronic Daily Intake mg/kg-d
Child		0.054	1	350	6	15	2190	0
Adult		0.054	1	350	24	70	8760	0
Adult+Child		0.054	1	350	30	70	10950	0

Direct Ingestion

$$CDI \text{ (mg/kg-d)} = \frac{C_{\text{fish}} \times IR_f \times FI \times EF_f \times ED}{BW \times AT}$$

Receptor	CW mg/L	IR <sub>f</sub> kg/day	FI	EF <sub>f</sub> days/year	ED year	BF L/kg	BW kg	AT years * day/year	Chronic Daily Intake (CDI) mg/kg-d
Child		0.054	1	350	6	5	15	2190	0
Adult		0.054	1	350	24	5	70	8760	0
Adult+Child		0.054	1	350	30	5	70	10950	0

Calculated from Surface Water Concentration

$$CDI \text{ (mg/kg-d)} = \frac{CW \times IR_f \times FI \times EF_f \times ED \times BF}{BW \times AT}$$

Variable		Value Used	Explanation/source
AT	Averaging Time	365 days/year x ED	Averaging time for noncarcinogens (EPA 1989a, 1991b)
BW	Body Weight	70kg (adult)	OSWER Directive, EPA 1991a
		15kg (child)	OSWER Directive, EPA 1991b
Cfish	Concentration in fish	mg/kg	
CW	Concentration in water	Chemical Specific	
BF	Bioaccumulation Factor	Chemical Specific 5.0 L/kg	5.0 L/kg, 90th percentile of study results in Appendix E
ED	Exposure Duration	6 years (child)	Two parts (child and adults) exposure for 30-years of exposure duration (OSWER Directive, EPA 1991b)
		24 years (adult)	
EFf	Fish Consumption Frequency	350 days/year	RAGS (EPA 1989a)
FI	Fraction Ingested	1 unitless	
IRf	Fish Consumption Rate	0.054 kg/day	OSWER Directive (EPA 1991b, 1995a)